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Original Communications

LATERAL CURVATURE OF THE SPINE FOLLOWING THORACOPLASTY IN CHILDREN*

MATHER CLEVELAND, M.D.
NEW YORK, N. Y.

THE inability to control cases of pulmonary tuberculosis with cavity formation and positive sputum and the extremely high mortality of over 60 per cent in the first year after admission to the hospital of this type of tuberculosis has led the medical and surgical staff of Sea View Hospital to the conclusion that radical measures are in the end conservative. All these patients who cannot be controlled by pneumothorax are given the benefit of complete surgical collapse of the lung. The results have been little short of amazing in this hopeless group of patients as a highly efficient operative technique has been devised. The vast majority of patients on whom this procedure has been tried have been adults. The result of the thoracoplastic operation in children, however, has been to produce a lateral curvature of the spine which has gone on to serious deformity. This problem has been presented to the orthopedic service of the hospital and merits our most thoughtful consideration.

The following six cases from the surgical services of the late Dr. John F. Honan and Dr. Pol. N. Coryllos are presented for study.

CASE 1.—G. C., 4416-33, an eleven-year-old girl, admitted to Sea View Hospital, April 2, 1931, with cough and hemoptysis. There was definite evidence of bilateral pulmonary tuberculosis clinically, and the roentgenogram showed cavitation in the left upper lobe. The sputum was repeatedly positive, Gaffky 2-6. Pneumothorax was attempted unsuccessfully.

*From the Orthopedic Service of Sea View and St. Luke's Hospitals.
Read before the American Academy of Orthopedic Surgeons, January 13, 1936.

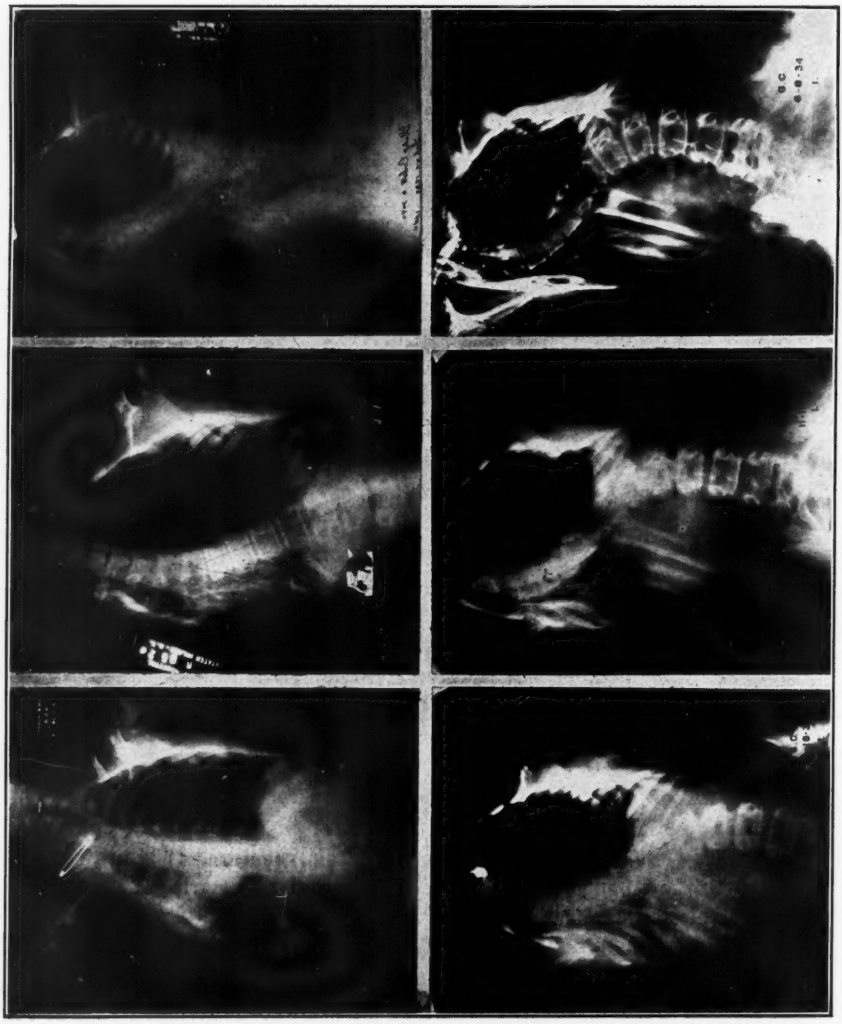

Fig. 1.

In May, 1932, a little over one year after admission, when the patient was twelve years old, a first-stage thoracoplasty was done, followed in three weeks by the second stage with total removal of the first two left ribs and part of the third to the tenth, inclusive.

The patient gained weight. After June 6, 1932, her sputum was negative, and her temperature remained below 100°. Within six weeks she was allowed up and around and was discharged from the hospital to the orthopedic service of St. Luke's Hospital in March, 1933, nine months after operation, well and with persistently negative sputum.

The history of her lateral curvature may be seen by studying the roentgenograms (Fig. 1).

On June 6, 1932, the day of her second-stage thoracoplasty, there could be seen a left dorsal curve of 14°, extending from dorsal 1 to dorsal 12, with the apex at dorsal 7.

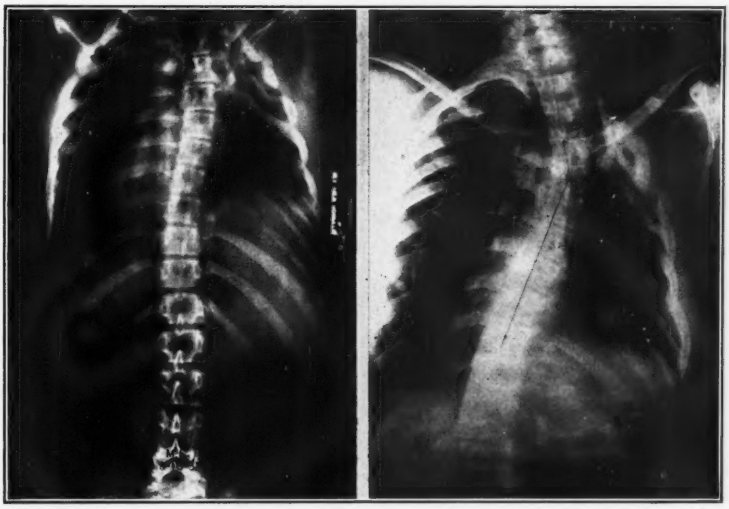

Fig. 2.

September 15, 1932, three months after operation, the curve had increased to 31°.

February 27, 1933, seven months after operation, the curve was 49° on weight bearing and 43° when the patient was lying down. At this point she was introduced to the orthopedic service.

April 18, 1933, nine months after operation, the curve was 53° on weight bearing and 43° when the patient was lying down.

November 11, 1933, seventeen months after operation, it was 64° on weight bearing and 60° when the patient was supine. At this point the patient was admitted to the orthopedic service of St. Luke's Hospital. No correction of the curve could be obtained. A celluloid jacket was made which the patient would not tolerate.

June 8, 1934, twenty-four months after operation, the curve was 73°, a fixed curve, as there was no difference between weight bearing and nonweight bearing.

CASE 2.—M. L., 6126-34, a girl of fourteen years, admitted to Sea View Hospital May 14, 1932, with pulmonary tuberculosis. The roentgenogram showed a cavity in the right upper lobe. In addition, she had a destructive arthritis of the left

hip joint without pain or spasm. This was tuberculosis of the hip joint, and the joint has subsequently been fused. Her sputum was positive on six occasions, Gaffky 2-6. Collapse therapy by pneumothorax was tried without success, and a right phrenicotomy was done, also without success.

In December, 1932, and January, 1933, first- and second-stage thoracoplasties were done, removing the right first to tenth ribs inclusive.

Two months after operation, with sputum negative and afebrile, the patient was discharged from the surgical to the medical service. She began to walk two months after operation, and shortly after this time, a lateral curvature was noted clinically.

The patient was discharged from the hospital in April, 1934, and reported to the orthopedic clinic of St. Luke's Hospital.

The progress of this patient's lateral curvature may be seen by a study of the roentgenograms (Fig. 2).

May, 1933, three months after operation, there was seen a right dorsal curve of 16°, extending from dorsal 1 to dorsal 10, with the apex at dorsal 5.

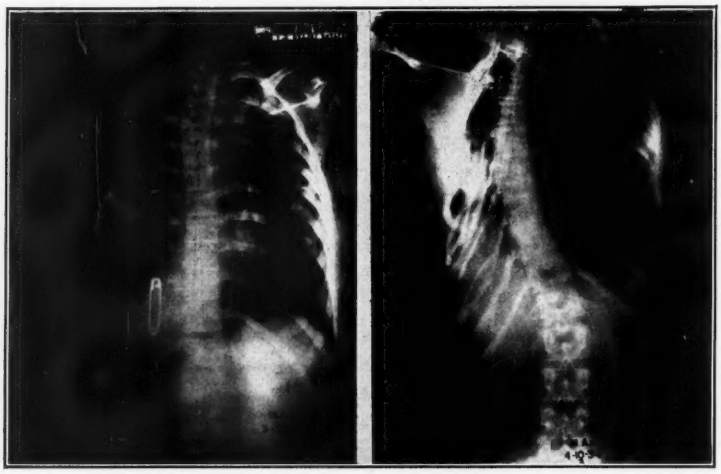

Fig. 3.

March, 1934, fourteen months after operation, this curve had increased to 33°.
August, 1934, nineteen months after operation, the curve was still 33°.

CASE 3.—M. A., 5663-34, a thirteen-year-old girl, admitted to Sea View Hospital February 29, 1932, with a history of loss of weight for three months. She had clinical evidence of disease in the left upper lobe, while the roentgenograms showed involvement of both lungs with several large cavities in the left lung. The sputum was positive, Gaffky 2-6. Pneumothorax was tried and the right lung improved. A left phrenicotomy was performed in January, 1933, and the sputum remained positive.

In February and March, 1933, a two-stage thoracoplasty was performed, with resection of the left first to twelfth ribs, inclusive.

The sputum became negative and temperature normal, and the patient was allowed up six weeks after the operation. In April, 1934, thirteen months after operation, the patient was discharged from the hospital and asked to report to the orthopedic clinic of St. Luke's Hospital, which she has failed to do.

The progress of her lateral curvature is revealed by a study of the roentgenograms (Fig. 3).

March 27, 1933, two days after the second-stage thoracoplasty, there was seen a left dorsal curve of 5°, extending from dorsal 2 to 11, with the apex at dorsal 6.

April, 1934, thirteen months after operation, just before the patient left the hospital, this curve had increased to 25°.

CASE 4.—E. L., 4087-33, a thirteen-year-old colored girl, admitted to Sea View Hospital February 2, 1931, complaining of cough, loss of appetite, and loss of 10 pounds in weight. The physical examination showed involvement of the left upper lobe, and the roentgenogram showed a cavity there. The sputum was repeatedly positive with high Gaffky for two and one-half years. Pneumothorax was unsuccessful.

In March, 1933, two years after admission, a left phrenicotomy was performed without benefit, and this was followed shortly by a two-stage thoracoplasty with

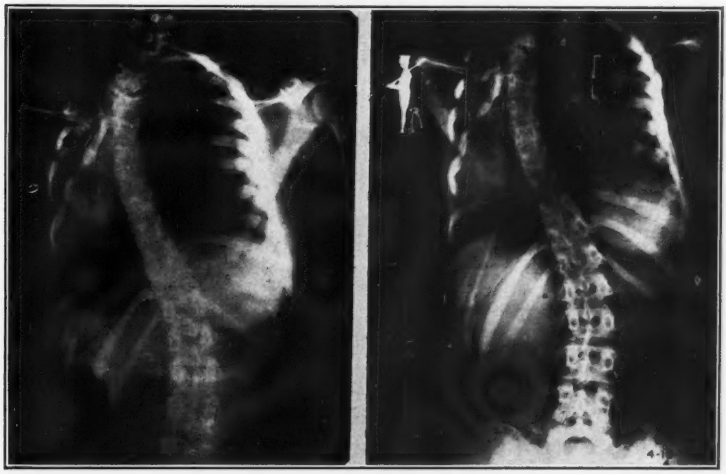

Fig. 4.

resection of the left first to tenth ribs inclusive. By July, 1933, the temperature was normal and the sputum was negative. The patient was allowed up four months after operation. In July, 1933, the presence of a lateral curvature was noted by roentgenogram.

The patient left the hospital in November, 1933, eight months after operation, against advice, with a small lesion in the right lung. She reported at the orthopedic clinic of St. Luke's Hospital in April, 1934.

The roentgenogram at that time (Fig. 4) showed a left dorsal curve extending from dorsal 1 to 12, with the apex at dorsal 6. This measured 30° on weight bearing and 20° with patient supine.

April, 1935, twenty-five months after operation, the roentgenogram showed the curve at 30° to 32° with the patient erect, no essential change.

CASE 5.—J. L., 8221-34, a white girl of fourteen years, admitted to Sea View Hospital April 28, 1933. She had been treated for three years in various hospitals and sanatoriums for tuberculosis and had recently had marked loss of weight. Her

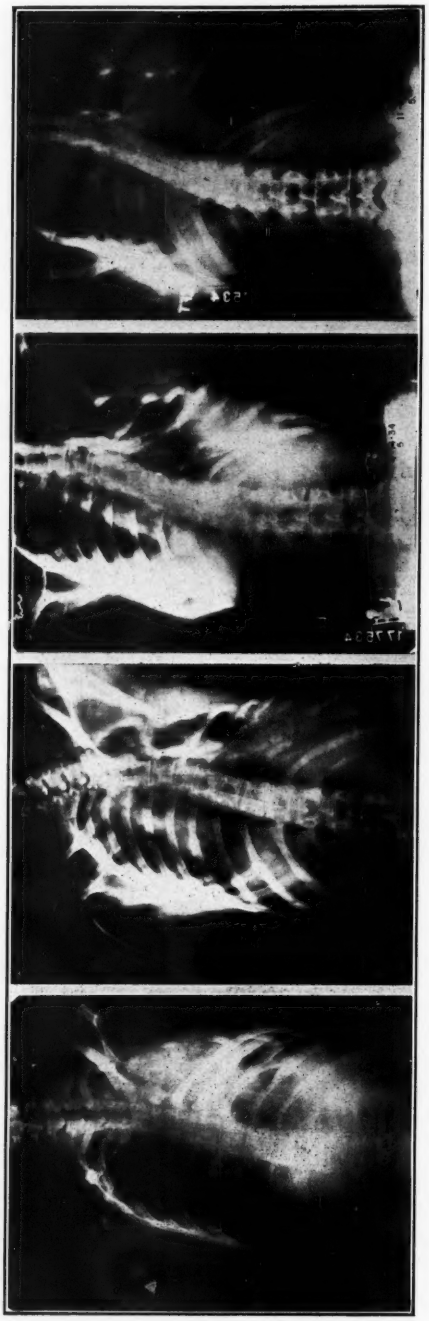

Fig. 5.

right lung showed extensive involvement clinically, and the roentgenogram showed caseous pneumonic tuberculosis in the right upper lobe with a large cavity. Sputum was positive, Gaffky 6, and gastric contents also showed tubercle bacilli. Three attempts at pneumothorax failed.

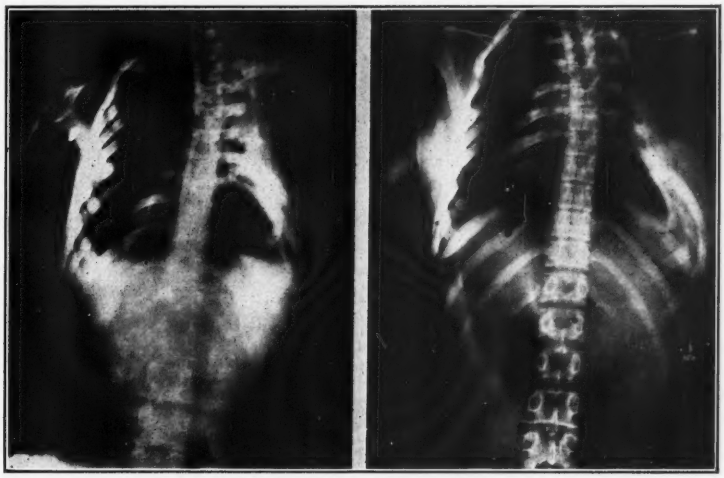

Fig. 6.

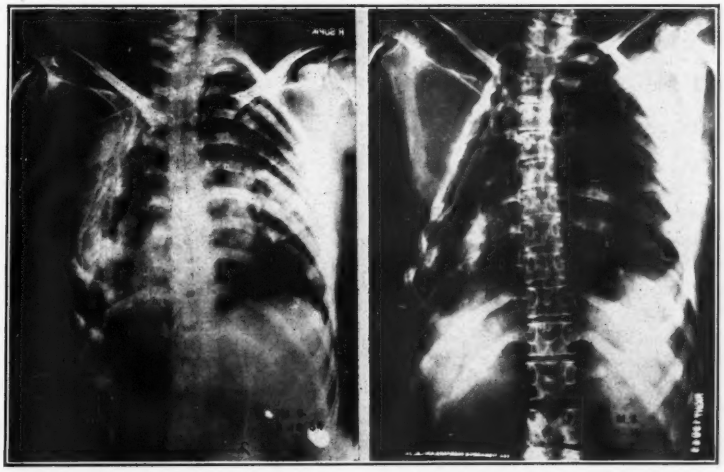

Fig. 7.

In June, 1933, the first- and second-stage thoracoplasties were done three weeks apart, and the right first to tenth ribs inclusive were removed. By three months after operation the patient began to show noticeable improvement, and by November, 1933, she had gained six pounds in weight, and her sputum was negative. At this time a lateral curvature was noted clinically.

In April, 1934, the patient was discharged from the hospital and reported at the orthopedic clinic of St. Luke's Hospital.

The progress of her lateral curvature is seen by referring to the roentgenograms (Fig. 5).

June 15, 1933, one week after the first-stage thoracoplasty, there was noted a right dorsal curve of 3°, extending from dorsal 3 to dorsal 11, with the apex at dorsal 6.

March 6, 1934, seven and one-half months after operation, this curve had increased to 14°.

April 14, 1934, nine months after operation, the curve was 16° on weight bearing and 10° with the patient supine.

November 8, 1934, seventeen months after operation, the curve was 17°.

CASE 6.—F. M., 9165-34, a white girl of thirteen years, admitted to Sea View Hospital October 30, 1933, with a three months' history of cough, expectoration, and loss of weight. The physical examination showed signs in the right upper lobe, and the roentgenogram showed a cavity there. The sputum was positive, Gaffky 3-6, and the temperature rose to 102° at times.

In December, 1933, and January 1934, a two-stage thoracoplasty was done, with resection of the right first to sixth ribs inclusive.

Sputum was negative after January 2, 1934, and the temperature remained flat. In February, 1934, a lateral curve was noted clinically. On April 14, 1934, the patient was discharged to the orthopedic clinic of St. Luke's Hospital but failed to report there.

The progress of her curvature may be seen by consulting the roentgenograms (Fig. 6).

February 5, 1934, one month after operation, there was seen a right dorsal curve, extending from dorsal 1 to dorsal 10, with the apex at dorsal 5. This measured 10° on weight bearing and 9° with the patient supine.

April 3, 1934, three months after operation, this curve had increased to 18° on weight bearing and 16° with the patient supine.

For comparison with these children, a typical adult case is added.

CASE 7.—M. S., a young woman of twenty-five years, with a left caseous pneumonic lesion and positive sputum. Pneumothorax was tried unsuccessfully for one and a half years.

In March, 1934, first- and second-stage left thoracoplasties were done, removing the left first to fifth ribs inclusive. In May, 1934, a third-stage thoracoplasty was done, and the sixth to ninth ribs inclusive were removed. Her sputum was negative after September, 1934.

This patient developed a lateral curvature of the spine which may be seen in her roentgenograms (Fig. 7).

In May, 1934, two weeks after operation, there was seen a left dorsal curve extending from dorsal 1 to dorsal 9, apex at dorsal 6. This was 12°.

In December, 1934, nine months after operation, this curve was 13° which represents no increase outside the range of error in measurement.

This patient simultaneously developed tuberculosis of her lumbar spine, and a fusion had been done.

COMMENT

1. *Type of Curve and Progress.*—These curvatures of the spine are all convex toward the side of the thoracoplasty. They resemble in this feature those curvatures seen in anterior poliomyelitis where the in-

trinsic muscles of the spine are paralyzed or weak on one side, and the unopposed action of the strong group of muscles produces a curve which is convex toward the weak side.

Another less likely factor which may play a minor rôle is the tremendous need for the remaining lung to expand in response to the individual's requirement for additional aerating surface. Various roentgenograms, especially those in Case 1, suggest this.

These patients have been observed only eighteen months for the average. One patient has been followed for three years.

The roentgenograms were not all taken at the same intervals, but they all tend to show a steady progress of the curvature to an average maximum of about 30° with one striking exception, the youngest child in the group, who reached a deformity of 74°.

TABLE I

PROGRESS OF LATERAL CURVATURE OF SPINE SHOWN BY ROENTGENOGRAM

TIME ELAPSED AFTER OPERATION		EXTENT OF CURVE IN DEGREES	AVERAGE
1 month	Case 1	14°	8°
	Case 3	5°	
	Case 5	35°	
	Case 6	10°	
3 months	Case 1	31°	21°
	Case 2	16°	
	Case 6	16°	
9 months	Case 1	53°	34.5°
	Case 5	16°	
12 months	Case 1	60°	33°
	Case 2	33°	
	Case 3	25°	
	Case 4	30°	
	Case 5	17°	
18 months	Case 1	64°	38°
	Case 2	33°	
	Case 5	17°	
24 months	Case 1	74°	53°
	Case 4	32°	

2. *Age.*—Aside from Case 1, these patients have been fourteen years old on the average and have, therefore, gained most of their growth. They are, in fact, almost adults. In these five patients the average curve at one year after operation is between 25° and 30°, which is about twice the amount of deformity that the adult case presented has shown. The one child of twelve years progressed rapidly during a two-year period until her curve has reached 74°, a horrible deformity.

A child with its most rapidly growing years ahead on whom a thoracoplasty is performed will almost certainly develop a serious and deforming curvature.

3. *Comparison of the Adult Case With the Children.*—The adult developed a curve of 12° to 13°, which is 20° less than the average of

the children. This deformity appeared about two weeks after operation, and 9 months later, there was no increase in the curvature. This curvature probably represents a minimal deformity which is incident to any complete thoracoplastic procedure. The fact that the children increase beyond this minimal deformity must be due to continued growth, and the amount of deformity to be expected will vary directly with the potential growth remaining in the spine.

4. *Treatment.*—The curvature begins at once while the patient is in bed. It is probably aggravated by weight bearing. It is doubtful if any extrinsic brace can be devised which these patients will tolerate, or which will be effective in checking the progress of the curve. The patients need the unhampered action of the remaining lung, and, therefore, they will not submit to a jacket, either of plaster of Paris or celluloid.

It is possible that a spine fusion from dorsal 1 to 12 inclusive would check the curve before it gets beyond 25°, which is not an unbearable deformity. These patients before their thoracoplastic operation are all in desperate circumstance with cavities in the lungs and positive sputum, and the surgeon would have to give grave consideration to the question of delaying the thoracic operation for two or three months while a spine fusion was performed. As a group, these patients with positive sputum are very undesirable operative risks. I am of the opinion, however, that if a thoracoplasty is necessary in a child, twelve years or younger, it should be preceded or accompanied by a spine fusion, done in multiple stages.

It is extremely regrettable that these thoracoplastic operations should have to be performed on children, but it may be granted that each of these patients was rescued from almost certain death. In each instance the sputum was rendered negative, and the temperature became normal. They have gained in weight and strength, and each has left the hospital. If possible, the thoracoplastic operation should be avoided in young children who have not reached their rapid growth period. It is almost certain that the younger the child, the greater will be the eventual deformity of the spine unless some effective means of checking the lateral curvature can be devised.

Some attention in the technique of thoracoplasty might well be directed toward investigating the fate of the posterior primary divisions of the dorsal nerves which innervate the intrinsic muscles of the spine.

DISCUSSION

DR. ARTHUR STEINDLER, Iowa City, Ia.—Dr. Cleveland has opened up a subject of considerable importance and one fraught with surprises. There is no doubt that one per cent of all scoliosis is tuberculous. I have had only eight cases of tuberculous scoliosis in over 800 cases. The reason the paper is interesting is that it shows the great contrast in the course of this scoliosis with thoracoplasty and

without thoracoplasty. It belongs in the same group with some of the spastic scoliosis cases. These cases are found almost invariably with the convexity toward the sound side. The clinical course is also mild. It depends entirely on whether the deformity is to one side or in the intrapleural region, with an emphysematous focus. The clinical course is occasionally rather acute, but on the whole, it is insidious, and it takes a considerable time for the scoliosis to reach this type of development. When thoracoplasty is performed, we see that the scoliosis is almost invariably on the affected side. It is very significant that it follows the curve to the affected side. I wish you could remember what has been done in years gone by in order to correct scoliosis by rib resection. At the time this method of treatment was first ventured, it was decided to do the resection on both sides. It is a good thing that it was not the sort of a thoracoplasty that will cause a shift of the spine to this side. When we had empyema, we noticed no increase in the curve to the sound side. We know that rhizotomy does not produce a curvature of the spine. Vischar, who followed 50 cases of rhizotomy, noted no curvature.

Take a patient with thoracoplasty; if the patient has had a curvature to the sound side, then the curvature will improve. If he had a curvature to the operated side, then it will grow worse. All in all, the question of healing of the spine after these very extensive operations is anything but clear, and I think it will require a good deal of study, particularly roentgenologic studies, to get at the trend of the curvature of the spine. We do not feel it is a true curvature. I believe, as Dr. Cleveland has pointed out, that the most appropriate treatment is to keep the patient on the side for a period of four months in order to prevent an increase of the disease. The paper has been most excellent.

DR. FREMONT A. CHANDLER, Chicago, Ill.—Dr. Cleveland has brought out a very interesting complication of thoracoplasty. We have to be a little careful that we do not condemn the thoracic surgeon for producing such a deformity. Pulmonary tuberculosis with cavity formation and positive sputum in children is almost a fatal disease. At the Municipal Sanatorium in Chicago, it is considered practically fatal; in older children, not quite so severe. It is in such a situation that the cooperation of the various specialists may accomplish something.

The situation with regard to thoracoplasty is new to me. I have taken a little time to go over the material to which I had access. In conjunction with this, I reviewed 280 cases of empyema. In these 280 cases of empyema followed by Dr. McHenry at Children's Memorial Hospital, we had but one persistent scoliosis. The others all had scoliosis to about 18° of curvature, and all showed the convexity away from the diseased or involved side. The maximum curve coincided with the height of their empyema process, as revealed by the x-ray films. These all cleared, in some with exercise and in some in which exercise was not an important factor. These healed by drainage.

In going over the thoracoplasties, I had access to the work which the late Dr. Hedblom and Dr. Van Hazel, who has now taken up his work, did. There were two observations in this group. Fortunately, the lower age limit for this group was seventeen years. Convexity was present in most of these cases toward the involved side prior to operation. It was very mild, however, and the shoulder was held high on that side. This may have an important bearing on the patient's subsequent condition. Following operation, which was done in multiple stages, the patients always rest on the operated side in attempt to splint it. The cause of the deformity may be due to a loss of muscle balance.

In empyema we may have an acute condition. In thoracoplasty we have a long wasting disease which must stand practically vulnerable to altered strength. The effect of such a curvature on a case of pulmonary tuberculosis is a loss of com-

pensatory curve in the remaining side. In addition, there is increasing fatigue caused by this curve. They need all the space they can have, except at the expense of marked deformity.

The spinal fusion suggested by Dr. Cleveland would probably be too severe for a child in the state of health and vitality that these children are in. There is one possibility which has not been touched upon as yet. I think it is evident that this curvature must be limited to procedure inside, that is, utilization of the resected rib. If the rib could be green-sticked and could produce a column of bone along the transverse processes, we might have some internal splinting on the side involved as the curve takes place with a marked increase in deformity.

DR. GEORGE J. GARCEAU, Indianapolis, Ind.—In discussion of Dr. Cleveland's paper, which is very timely, I should like to emphasize some of his points and also to make some suggestions as to treatment.

First, age. Most of these patients who develop scoliosis are under fourteen years of age. None of the adults develop scoliosis of any severity. That is because of the rapidity of growth between twelve and fourteen years. In thoracoplasty done after fourteen or fifteen, we get little scoliosis. It seems to me that a study of Dr. Cleveland's cases reveals the mechanics of the scoliosis. He did not go very deeply into that phase; therefore, I would like to say a few words about it. The force expended upon the ribs on one side is the primary cause of the scoliosis. You remove the ribs on one side, and the patient breathes with the good lung. This procedure is expended on the ribs by the process of expansion of the wound, but all the force is expended at the costovertebral joint, causing a rotation of the vertebrae, as well as a sagging of the vertebrae toward the affected side. In those cases which he presented, the increase in the scoliosis ceased after eight or ten months. That is because at that time the periosteum has elevated most of these ribs, and we now have a more equal force on both sides expended by the ribs. In the Schede type of thoracoplasty, where the periosteum is removed and the ribs do not regenerate, the increase in the scoliosis is marked, even after ten months, unless the ribs are held in place. This force on the ribs is augmented by the muscle balance, and especially is that true in the Schede operation.

As to treatment, I should like to make a few suggestions. The patient should be caused to lie on the well side, with the arm abducted, and a wedge placed below the apex of the curve. It will develop a curvature much less rapidly if this is done. This should be done until such a time as a brace can be made. We make a modification of the Risser type of jacket. I have not tried plaster jackets.

As to fusion, I do not know. I would certainly hesitate about fusion from the sixth to the twelfth dorsal vertebrae in children as sick as they are. The fact is, we should not criticize the thoracic surgeon, but should try to cooperate with him and try to provide some treatment that will prevent scoliosis from developing, because most of these children die.

Dr. JOHN R. MOORE, Philadelphia, Pa.—Dr. Cleveland has presented the tragedies associated with this particular type of scoliosis. Of this scoliosis in children, I have had few cases, and these have very well followed out Dr. Cleveland's conclusions. I thought it would be of interest to collect a number of cases of thoracoplasty in the adult, cases that were at least five to ten years postoperative, for the purpose of making an absolute comparison between the growing and nongrowing period.

Fifteen cases were collected from the Eagleville Sanatorium, Norristown, Pa., through the courtesy of Dr. Louis Cohen. These were adults upon whom thoracoplasty had been performed, between the ages of twenty-one and thirty-five. The average duration of the thoracoplasty was from five to ten years. In all these cases

thoracoplasty had been performed for tuberculous lesions of one side. In none of these cases did the curve amount to more than 20°. It was entirely insignificant clinically, and afforded only passing comment from the roentgenologist. The rotation associated with the usual lateral deformity was insignificant, and of only slight degree, and could scarcely be determined even on very close inspection. We should conclude from this small group of cases of five to ten years' duration postthoracoplasty, that thoracoplasty in the adult is accompanied by scoliosis of insignificant importance. Comparing this with scoliosis associated with thoracoplasty in children, one can only draw theoretical conclusions.

First of all, the contention that the need of a compensatory enlargement of the lung demanded or compelled the curve is somewhat questionable.

The curve in adults was seen usually about the sixth week postoperative, and it showed rarely any increase over the degree present at that time. We must assume that the scoliosis seen in children is associated with, and possibly secondary to, (1) growth, such as we see in all our scoliosis in children, and (2) possibly the relative length of the thoracoplastic side. Another point of some interest is the fact that in the adult prior to the thoracoplasty in all these cases in which the lesion predominated on one side, the convexity of the curve was away from the site of the lesion. After thoracoplasty, this curve was reversed. One might think that the mild curve seen occasionally in the adult in the dorsal region represents a very mild and temporary arrest of growth in this position in early life.

DR. MATHER CLEVELAND, New York, N. Y. (closing).—There are just one or two points that I should like to emphasize that have been stressed by those men who have been kind enough to discuss this paper.

Dr. Chandler said, and I said it too, that each of these children has been wrested from certain death at the price of a curvature of the spine. In only one instance was this curvature of a deforming nature. In other instances, the curve of 25° was not unbearable.

These patients, in answer to Dr. Steindler, should be kept in bed longer. I will agree with that. These children were not transferred to the orthopedic department after discharge from the surgical department. They were allowed up far too early. If you will recall some of these roentgenograms I showed, however, the curvature had already started while the children were in bed and increased quite markedly while in bed. I think weight bearing unquestionably had some effect in addition. The earliest x-ray that I have is three days after operation, and the curve at that time was only three degrees. We had seen the x-rays of these patients before operation, and none of these had an appreciable curve.

Dr. Chandler's suggestion that the ribs might be turned down to form a splint is a very good one, and I am going to refer it to our thoracic surgeons.

Dr. Garceau made the statement that most of these curves had reached their maximum at about the fourteenth year. I do not think that is correct. I think they have reached their maximum at the time the child has stopped growing. Most of these children were about fourteen years of age, and they probably had little growth left in the spine. In one instance, a child of twelve, the curve had increased over two and one-half years until she was fourteen, and it was there that her growth stopped.

DR. POL. N. CORYLLOS, New York, N. Y. (comments in letter).—I read with pleasure your paper. I think that it deals in a clear and concise manner with a very important complication which frequently follows thoracoplasty in children.

There are a few remarks which I would like to add:

First, all children, even below the age of fourteen, do not develop scoliosis. Offhand I remember one case, a girl of fourteen years, who had an extensive thora-

coplasty without the slightest degree of scoliosis. On the contrary, marked scoliosis developed in patients over twenty and thirty years of age. It seems that patients who have remained in bed for four months or longer after operation are less apt to develop spine deformities. This may be due to regeneration of bone which acts as a fusion of the spine.

Regarding the mechanism of this scoliosis, I think that your first conception is correct. The mechanism is similar to paralytic scoliosis. The vertebrae through the ribs are submitted to the action of the intercostal muscles which tend to bring the ribs together. When ribs are resected on one side only, the action of the intercostals upon the ribs of the healthy side is no longer counterbalanced; therefore, the whole column tends to incurvate toward the opposite side.

I do not think that the vicarious hypertrophy of the healthy lung has any influence whatsoever upon the production of this type of scoliosis, because, if anything, the widening of the intercostal spaces of the healthy lung would tend to produce a scoliosis with convexity toward the healthy side.

One other remark I would like to make. In one case which I have operated upon I made a greenstick fracture of the transverse process as suggested by Dr. Chandler. This patient has not shown any scoliosis. I cannot say whether it is due to the procedure or not.

SKELETAL DEFORMITIES IN CHILDREN RESULTING FROM EMPYEMA AND METHODS OF PREVENTION*

J. DEWEY BISGARD, M.D.
OMAHA, NEB.

THE completely successful treatment of any disease implies, not only the cure of that disease, but also the restoration of the patient's health to as nearly a normal state as possible. It demands anticipation and recognition of complications and sequelae, and the application of measures for their prevention or treatment. Upon the surgeon who treats empyema and other forms of thoracic disease, it imposes the responsibility for the prevention of such complications as deformities of the thoracic skeleton. This is his problem, I believe, because the deformities have their inception while the patients are under his supervision, because they are almost invariably preventable, and because the time to cure them is in their incipiency or before they occur.

Severe deformities are in most instances indictments of inadequate treatment because they are preventable. They are important, although they occur infrequently, because they are irreparable and because the extensive curvatures of the spine and distortions of the costal framework produce not only disfigurement of great concern to the patient, but also incapacity from impairment of cardiac and respiratory functions which not infrequently results in premature death.

In a previous publication[1] there was reported an investigation of this subject based upon a study of 518 patients with various forms of thoracic disease and 336 patients operated upon for thoracic disease. The study was concerned primarily with an analysis of the factors concerned in the production of deformities, the types of deformities produced, their frequency and clinical significance. It was found that while minor lateral deviations of the thoracic spine had developed in a large percentage of cases, in only eleven cases were the curvatures great enough to give rise to a deformity of major clinical importance. Eight of the eleven had developed in cases with chronic pleuritis (seven of them empyema), and the others in cases in which successful treatment had necessitated an extensive Schede thoracoplasty or lobectomy. Significant was the fact that these deformities had developed only in children and young adults (less than twenty years of age).

It was apparent that all thoracogenic spinal curvatures are two types, those associated with pleuritis (mainly empyema) designated as pleurogenic scolioses, and second, those resulting from the removal of ribs, or thoracoplasty scolioses.

*From the Department of Surgery, University of Nebraska School of Medicine.
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I. PLEUROGENIC SCOLIOSIS

In the literature, pleurogenic scoliosis is referred to as empyema scoliosis. The term "pleurogenic scoliosis" will be used because it describes more accurately the anatomic relationship and because the curvatures often are produced by pleural disease other than empyema, such as simple serous effusions.

To understand the mechanism of production of thoracogenic curvatures, it is necessary to remember that normally the spine is maintained in a state of static equilibrium by a balance of opposing forces which act on the two sides of the vertebral bodies. These forces are exerted not only by ribs, muscles, and other soft tissues of the chest wall, but

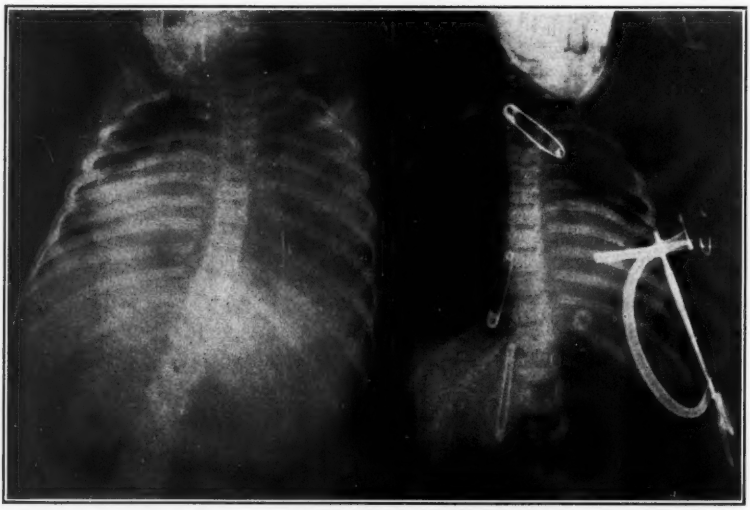


Fig. 1.—*A*, an acute empyema of the left side with a massive effusion which has displaced the mediastinum far into the right side and diminished the available aerating lung to such an extent that the patient was cyanotic and dyspneic. Note the widening of the intercostal spaces on the left side and the spinal deviation with the convexity to the left. This is an unusual type of curvature seen only in presence of massive effusion. *B*, the same patient after drainage was established. Note the narrowing of the left intercostal spaces and the reversal of the spinal curvature, which is now of the spastic type characteristic of acute empyema. (Bisgard, J. D.; Arch. Surg. 29: 417, 1934.)

also by pressures and stresses which arise within the thoracic cage. With the onset of empyema there develops, as a rule, a slight lateral deviation of the spine with the concavity on the affected side. (Fig. 1 *B*.) This results from contracture of the muscles of that side as a splinting mechanism comparable to the spasm of the muscles of the abdominal wall in cases of peritonitis. With early cure, the spasm is released and the curvature corrects spontaneously. If the disease becomes chronic or is productive of much pleural scar, a second and more important deforming mechanism develops. Like all scar tissue, the pleural scar

eventually contracts and through adherence to the ribs pulls them centripetally. Since ribs, like bucket handles, can pivot only at their extremities (the vertebral articulations posteriorly and the cartilages anteriorly) and inscribe arcs as they are drawn mesiad, they also move downward. This force is transmitted to the vertebral bodies through their costovertebral articulations, traction being exerted on the uppermost thoracic vertebra by the upper ribs which are pulled downward, and pressure being exerted on the lower ones, especially on those

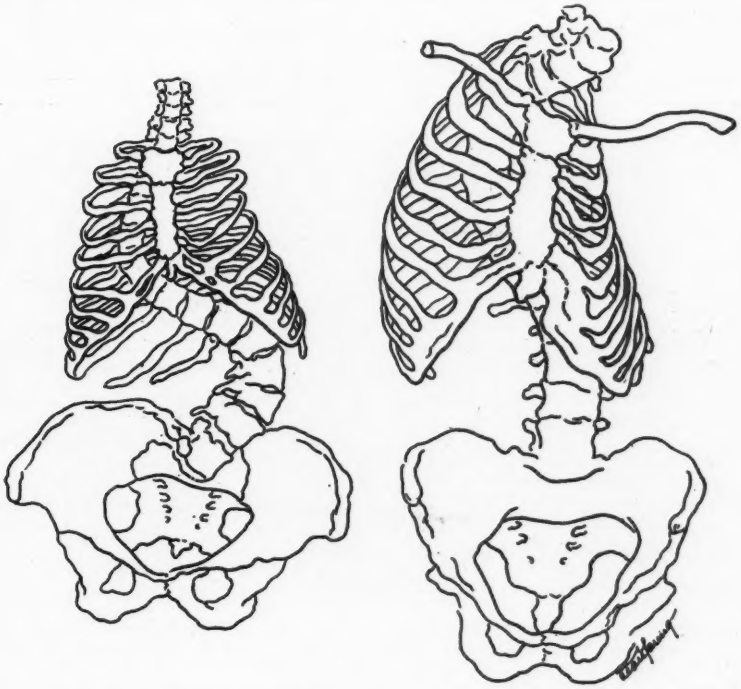


Fig. 2.—Diagrammatic drawings illustrating Table I, for a comparative analysis of rotary and nonrotary scoliosis. To the left is illustrated the rotation deformity of the thorax of idiopathic scoliosis, which typifies the rotary type of scoliosis. The drawing to the right represents the deformity of pleural scoliosis which uniquely is a nonrotary type of curvature (Table I). (Bisgard, J. D.: Arch. Surg. 29: 417, 1934.)

at the apex of the curve. The result is a spinal deviation with the convexity of the curve projecting into the healthy side (Figs. 2 and 6), directed reversely to that produced by thoracoplasty.

The nature of the etiology of pleural scoliosis makes it a distinct clinical entity. It differs from all of the other types of scoliosis, in that there is no, or only slight, rotation of the vertebral bodies. It is uniquely a nonrotary curvature. This disobedience to the well-recognized physical law (stressed by Lovett[2]) of the dissipation of part of the bending

force of rods into the production of rotation or twisting is due to the immobilization of the ribs in their retracted positions. The vertebrae cannot rotate against the resisting pressure exerted on them by the ribs, which are rigidly fixed by the pleural scar. This scar, in certain instances, undoubtedly exerts force directly on the vertebral bodies, which helps to prevent rotation and usually also fixes the mediastinum to the spine so that it deviates with the spine.

The retraction of the thoracic wall by scar tissue restricts the respiratory excursions and greatly reduces the volume capacity of the affected hemithorax.

TABLE I

NONROTARY SCOLIOSIS	ROTARY SCOLIOSIS
Pleural Scoliosis	*Idiopathic Scoliosis*
1. On the side of the concavity	1. On the side of the concavity
(a) Increased downward inclination of the ribs; pulled downward and mesiad by pleural scar.	(a) Abnormally horizontal position of ribs; since the ribs rotate with the vertebrae toward the side of the convexity, their arched shapes cause them to lift upward.
(b) Intercostal spaces narrowed; ribs contact or overlap and fuse.	(b) Intercostal spaces unchanged or only slightly narrowed.
(c) Thoracic wall (costal arches) flattened anteriorly and posteriorly.	(c) Thoracic wall (costal arches) flattened posteriorly but bulged anteriorly, owing to vertebral rotation.
(d) As a result of the retraction of the ribs, hemithorax smaller than that on the opposite or convex side; the circumference from 1¾ to 3 inches (3.5 to 7.62 cm.) smaller in five cases measured.	(d) As ribs rotate upward and outward this hemithorax becomes larger than that of the opposite or the convex side.
(e) Lung on this side relaxed and atelectatic.	(e) Lung on this side partially emphysematous and partially atelectatic.
2. On the side of the convexity	2. On the side of the convexity
(a) No posterior bulge of thoracic wall because there is no rotation deformity.	(a) Thoracic wall (costal arches) flattened anteriorly and bulged posteriorly at apex of curve, owing to rotation of vertebrae.
(b) Shoulder high but scapula not prominent.	(b) Shoulder high and scapula prominent.
3. Either no, or only slight, rotation and structural change of vertebral bodies.	3. Always rotation and often great; usually structural changes (wedging).
4. Curves usually rigid and often short and sharply angulated.	4. Frequently not rigid and often long and gently arched.
5. Often compensatory curvatures do not develop until the patient sits up, and the spine bears weight.	5. Patients are ambulatory, so compensatory curvatures frequently develop simultaneously with the primary curves.
6. Mediastinum usually deviates with the spine, to which it is fixed by scar tissue.	6. Mediastinum does not deviate; it is free and maintains its normal position.
7. Reduction of vital capacity proportional to deformity. (Flagstad and Kollman: J. Bone & Joint Surg. 10: 724, 1928.)	7. Same.

To determine the influence of age of the patient and of the duration of the empyema upon the incidence and severity of the deformities,

there were studied 102 cases of acute empyema and an equal number with chronic empyema. Both the incidence and the extent of the spinal curvatures varied roughly in inverse proportion to the age of the patients, and in direct proportion to the duration of the disease; that is, the younger the individual, the more likely and the more extensive the curvature; and the more chronic the empyema, the more likely and the more extensive the curvature.

II. THORACOPLASTY SCOLIOSIS

This type of scoliosis differs from pleurogenic scoliosis which is nonrotary and resembles all other types of scoliosis in that the vertebral bodies and the costal framework undergo rotation as the spine bends

Fig. 3.—Roentgenograms in a case of *A*, idiopathic scoliosis, and *B* pleural scoliosis. Compare with Fig. 2 and Table I. Note that the mediastinum did not deviate with the spine in *A* but did deviate in *B*. (Bisgard, J. D.: Arch. Surg. 29: 417, 1934.)

laterally. The widely different clinical pictures of pleurogenic and thoracoplasty scoliosis are presented in Table I and Figs. 2 and 3.

Thoracoplasty scolioses result from the imbalance of the spine produced by removal from one side the supporting struts, the ribs. These are the principal agents for the support of the thoracic vertebrae by virtue of their rigidity not only as bony braces, but also because of their service as instruments through which the forces exerted by the attached muscles and ligaments and the forces of intrathoracic pressures and stresses are transmitted to the vertebrae through their costovertebral articulations. Since the bony framework of the thorax is made up of a series of superimposed semirigid circular units, forces acting on one area are transmitted throughout the circumference of each unit. Normally,

each pair of ribs grips with pressure the vertebra with which it articulates. If this pressure is unequal on the two sides, or if it has been removed from one side by the extensive resection of ribs, the unopposed ribs push the vertebrae toward the unsupported side, and a deviation results. A spine which was straight before operation will deviate with the convexity of the curve projecting into the side on which the operation was performed (Figs. 4 *A* and *B*).

A study of 131 patients treated by thoracoplasty showed that the contour of the spine is always altered, but this alteration is influenced by the preoperative contour.

1. A spine which was straight previous to operation almost invariably deviated with the convexity toward the side on which operation was performed. This occurred in 59 (45 per cent) of the cases.

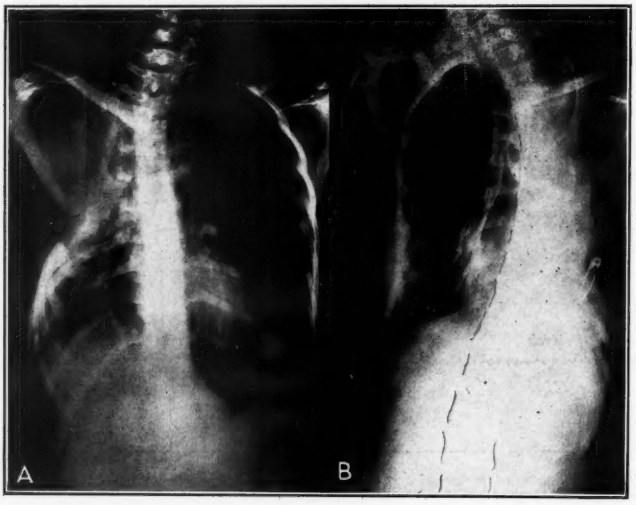


Fig. 4.—Thoracoplasty scolioses. *A*, a short, high dorsal dexter convex lateral curvature has developed in a previously straight spine as a result of the imbalance produced by the partial extrapleural posterolateral thoracoplasty. As in this case, most of the spinal deviations which develop with the extrapleural type of thoracoplasty in adults are so slight that they have little clinical importance. *B*, a retouched roentgenogram in a case of chronic empyema in which the patient was cured by thoracoplasty of the Schede type. Almost total costectomies of the third to the ninth ribs, inclusive, were done. A deforming curvature has been produced by the gross imbalance resulting not only from the extensive costal resections, but also from the wide excision of muscles and other soft tissues of the thoracic wall and of the restraining pleural scar. Before operation, the spine deviated in the opposite direction. A pleural scoliosis, therefore, has been overcorrected and converted into a thoracoplasty scoliosis. (Bisgard, J. D.; Arch. Surg. 29: 417, 1934.)

2. If it deviated with the convexity toward that side before operation, the curvature was greatly exaggerated by thoracoplasty. This occurred in 21 cases (16 per cent).

3. If, however, it curved in the opposite direction (preoperative pleural scoliosis), the deviation was partially or completely corrected. This occurred in 12 (9 per cent) of the cases. Often the contour was overcorrected; this occurred in 39 (29 per cent) of the cases. See Fig.

5 *A*, *B*, and *C*. In 5 cases, a double or S-shaped curve developed, composed of a thoracoplasty curvature superimposed on a preexisting pleural scoliosis.

Of the 131 thoracoplasties, 101 were extrapleural and were carried out in the treatment of pulmonary tuberculosis. The remaining 30

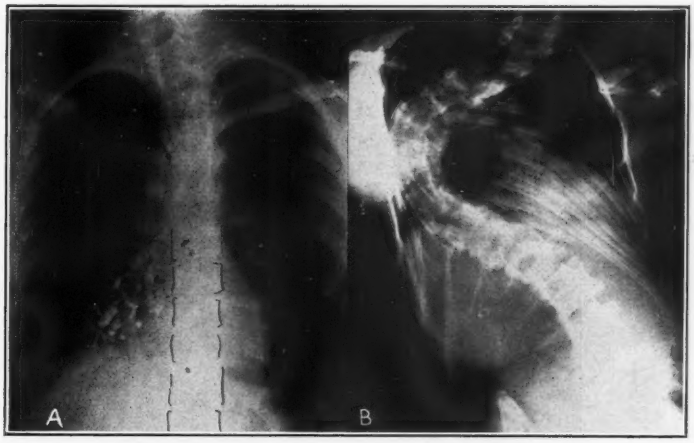


Fig. 5.—The influence of thoracic operations on the spine. *A*, roentgenogram of the patient with bronchiectasis of the right lower lobe, which has been filled with iodized poppy-seed oil, 40 per cent. Note the slight sinister convex scoliosis. *B*, the same patient three years after total excision of the right lower lobe after removing long segments of three ribs. The spine has reversed its curvature and completely collapsed into the side on which operation occurred. Note the rotation of the vertebrae and the deformity of the thoracic wall caused by the rotation. *C*, photograph of the same patient illustrating the characteristic deformity of rotary scoliosis. (Bisgard, J. D.: Arch. Surg. 29: 417, 1934.)

operations were Schede thoracoplasties for empyema; the intercostal soft tissues and much of the parietal pleural scar were excised as well

as the ribs. As a result of this additional resection (soft tissues and restraining parietal pleura), the Schede type of operation, as a rule, produced greater imbalance and greater alteration of spinal contour than did the extrapleural type. Compare Figs. 4 *A* and *B*.

PREVENTION AND TREATMENT OF PLEUROGENIC SCOLIOSIS

Although the principal interest in most of the spinal deviations incident to thoracic disease and thoracic operations is academic, a certain small minority progress to a stage of deformity. The deformities occur only in certain cases of pleural scoliosis, occasionally subsequent to extensive costal resections and lobectomies, and only in children and young adults.

Fig. 6.—Case 1.—Pleurogenic curvature in a boy, aged sixteen, resulting from chronic empyema at age two. The deformity caused his death. (See Case 1 in text.)

As stated previously, extensive deformity from any cause produces not only disfigurement, but also much impairment of cardiac and respiratory functions and often subjective symptoms, such as pain and ease of fatigue. The restriction of respiratory excursions greatly reduces the capacity of pulmonary ventilation. Disturbed anatomic relations of the heart and great vessels which impair their functional efficiency and evidence of interference with the return flow of the pulmonary circulation with chronic passive congestion of the lungs have been observed frequently at necropsy (Eckhardt[3]). These factors not only limit the patient's activities but also diminish his ability to combat any severe illness successfully. Pneumonia is a frequent cause of death.

A case history illustrating progressive incapacity terminating with death as a result of the deformity produced by empyema in infancy follows:

CASE 1.—M. F., a white boy, aged sixteen, entered the University Hospital on September 12, 1935, and died twelve hours later from progressive dyspnea and cyanosis. At two years of age, he had a postpneumonic empyema of the left chest which was drained by intercostal incision two months after onset and after it had become an empyema necessitatis. Two months later a segment of the eighth rib was removed. Drainage continued for one year, and before it had ceased the mother states that the child had a deforming curvature of the spine. The deformity gradually became more extensive and for four years, between the ages of six and ten, he was treated without benefit by plaster jackets and body braces. He remained well except that he was unable to play with other children because exertion brought on extreme dyspnea. His activity became more and more limited, and during the last six months he had been compelled to spend much of his time in bed. Even at bed rest he had paroxysms of dyspnea and cyanosis, such as the one presented upon admission to the hospital.

Examination showed extreme dyspnea and marked cyanosis in a poorly developed and poorly nourished boy, with an extensive spinal curvature and deformity of the chest wall of the pleurogenic type (Fig. 6). He was afebrile, had an enlarged heart and a rapid feeble pulse. Both physical and x-ray findings of the pulmonary fields showed only emphysema of the right lung and some compression of the left at the base.

The postmortem examination revealed slight emphysema of the right lung, a small compressed left lung, and extensive distortion of the great vessels and marked hypertrophy and dilatation of the right side of the heart. No other cause of death could be found except for the mechanical disturbance to the lesser circulation brought about by the deformity.

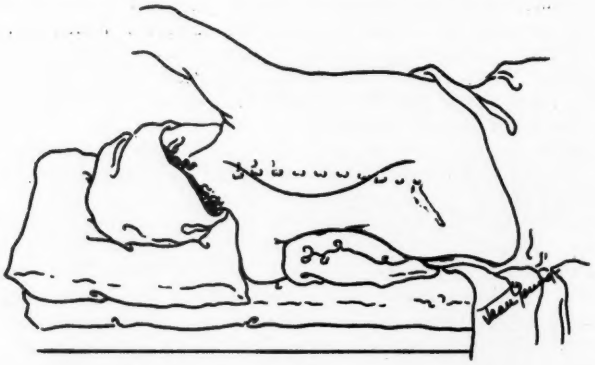
As pointed out previously, the spinal deviations associated with acute nontuberculous empyema usually correct themselves when the empyema cavity is obliterated promptly. Deformities develop with the chronicity of the disease, which, with few exceptions, reflect improper treatment. The first objective in the prevention of pleural scoliosis is, therefore, the early closure of empyema cavities by early, adequate, and dependent drainage. Von Kolliker[4] stated that the development of scoliosis is best prevented by the rapid healing of the disease. No scoliosis developed in any of his cases in which drainage was done early by rib resection.

In pure tuberculous effusions, rapid closure of pleural cavities may be effected only by repeated thoracenteses, because drainage is contraindicated, but drainage is applicable to many tuberculous effusions which are secondarily infected by pyogenic microorganisms.

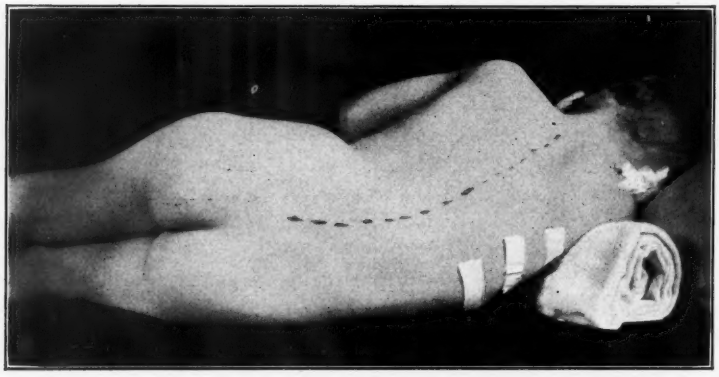
In all cases with pleural effusions, regardless of the etiology, a constant effort should be made to hold the spine in alignment or slightly overcorrected during the major portion of each twenty-four hours, not only during the period of active treatment, but also for several months after the pleural cavity has been obliterated, in order to prevent deformity from latent contracture of the pleural scar. With the exception of infants and uncooperative children, this can be accomplished in

most patients by posture; the patient should lie on his side in such a way as to deviate the spine in the direction of overcorrection sufficiently to counteract the tendency to a pleural scoliosis.

The spine can be controlled and held in a corrected or overcorrected position most simply and most effectively by posture alone, or by bending the spine by postural wedging.[5] In the latter procedure, illustrated in Figs. 7 and 8, the patient lies on either side on a soft compression



A.



B.

Fig. 7.—Postural wedging. The patient lies on the diseased side, or the side on which operation was performed, over a pillow which has been rolled in the direction of its long axis and placed at right angles to the axis of the body. By placing this fulcrum properly, the spine can be wedged in the direction desired. To correct and prevent pleural scoliosis, the fulcrum is placed high in the axilla, wedging the spine in the direction of overcorrection. To correct a thoracoplasty scoliosis, the wedge is placed slightly more distal, that is, directly opposite or slightly caudal to the apex of the curve. *A*, the change of spinal contour which can be brought about by this procedure is illustrated diagrammatically by the straight alignment of the spinous processes. The curved, broken line illustrates the position of the spine of a thoracoplasty scoliosis before wedging. *B*, empyema of the right pleural cavity. The spine has been wedged into overcorrection with the fulcrum placed high in the right axilla.

wedge, such as a pillow, which is rolled in the direction of the long axis and placed at right angles to the axis of the body. If this fulcrum is

placed properly, the weight of the body wedges the spine in the direction desired. In cases of developed or potential pleural scoliosis, the wedge is placed high in the axilla of the diseased side (with the arm completely abducted and the head elevated) so that the entire thoracic spine is deviated in the reverse direction, the direction of overcorrection. If the empyema cavity is large, it is desirable to have the patient lie upon the diseased side (Fig. 7) because it permits dependent drainage, immo-

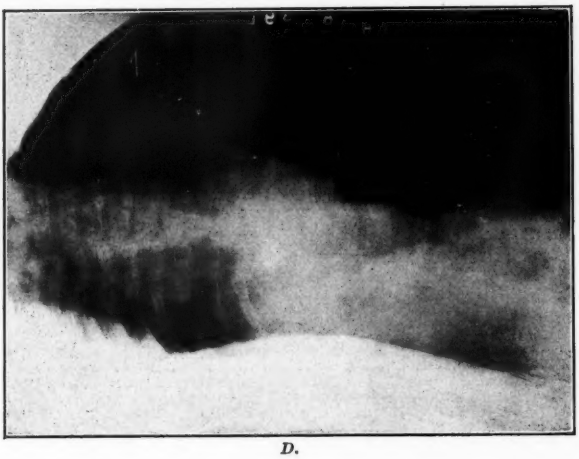
A.

B.

Fig. 8.—Postural wedging. An infant three months old with empyema of the right pleural cavity. Spine held constantly in overcorrection in a plaster of Paris bed with a removable restraining lid. Roentgenograms of infant show curvature and its overcorrection.

bilizes that side, and does not interfere with the free respiratory excursions and pulmonary ventilation of the healthy side. This position can be maintained (whereas it might otherwise be impossible on account of pain) by anesthetizing the wound by crushing three intercostal nerves at the time of establishing drainage. It has been my practice to establish drainage by costal resection through the vertical incision advocated by Churchill. This not only has the advantage pointed out by Churchill,

C.



D.

Fig. 8.

but permits easy exposure of one intercostal nerve above and one below the resected costal segment. These three nerves I routinely crush.

If lying upon the affected side is intolerable, postural wedging can be accomplished equally or more effectively with the patient lying on the unaffected side with the fulcrum placed at the apex of the curve as illustrated in Fig. 8.

If the curvature persists after the patient becomes ambulatory, correction can be accomplished and maintained by the use of a special type of plaster jacket. It is applied with the body bent to the affected side until the lumbar, or secondary, curvature has been corrected. The cast extends slightly below the greater trochanters on both sides and to the twelfth rib on the unaffected side. Very little padding is used, and

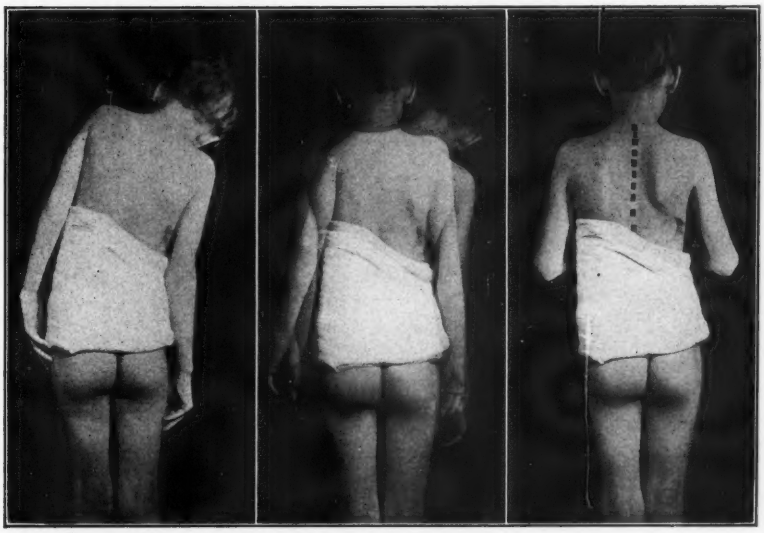


Fig. 9.—Plaster body cast used in ambulatory treatment. It is applied with the thoracic, or primary, curvature exaggerated so that the spine is wedged straight upon assuming an erect posture.

the cast is made to fit very snugly. As the patient assumes an erect posture to establish balance, he automatically corrects the thoracic or primary curvature by wedging the spine over the edge of the cast. The principle is illustrated in Fig. 9. This type of jacket should be worn for two or more months. After its removal, the patient should be seen at monthly intervals for at least a year, and with the slightest evidence of a recurrence of the curvature, the jacket or cast should be reapplied.

Young children may be held in position for postural wedging in a plaster bed which is molded to the body with the patient lying on the side in a position of overcorrection. The side walls should be high enough to prevent the child from rolling out of this position. A plaster

of Paris bed is illustrated in Fig. 8. Overcorrection in such a bed should be maintained for many weeks and until there is no tendency to recurrence of the curvature.

PREVENTION AND TREATMENT OF THORACOPLASTY SCOLIOSIS

Although the vertebral column deviates or sags into the unsupported side of the chest after the extensive costal resections of a posterolateral thoracoplasty, it is sufficiently plastic to permit considerable molding and correction by force until it becomes fixed from four to eight weeks after operation by regenerated ribs, organized fibrous tissue and reinserted muscles. If correction is maintained during this reparative period, permanent curvatures will be minimized and often prevented. If, how-

Fig. 10.—*A*, a tuberculous empyema of two years' duration in a child of four years has been productive of a moderately severe pleural scoliosis. *B*, the same patient two weeks after completion of a Schede thoracoplasty. Note the correction of the pleural scoliosis by the imbalance produced by the operation. (Bisgard, J. D.: Arch. Surg. 29: 417, 1934.)

ever, the periosteum has been destroyed or excised (as it is in a Schede thoracoplasty), the resected costal segments are not replaced, and the spine is permanently deprived of much of its support on the side on which operation has been performed. In many instances, however, this lack of support may compensate for the previous imbalance from the pleural scar and merely correct a preoperative pleurogenic scoliosis. Thus, as illustrated in Fig. 10, the operation performed for the purpose of obliterating a chronic empyema cavity often incidentally corrects the curvature which is usually present in these cases. Not infrequently it overcorrects and replaces the pleurogenic by a thoracoplasty type of scoliosis. The postoperative prophylactic maintenance of correction by postural wedging in these cases is beneficial but less effective than in the

cases in which the extrapleural type of operation has been done. It is desirable, therefore, to obliterate as much of the empyema cavity as is possible by extrapleural resection of ribs.

To prevent and correct thoracoplasty scoliosis, postural wedging should be instituted within a few days after operation and maintained for at least six weeks, or until the reparative processes have fixed the thoracic wall. The procedure is carried out with the patient lying on the operated side but with the wedge placed exactly opposite, or slightly caudal to, the apex of the curve, as illustrated in Fig. 7 *B*.

With cooperation from the patient, postural wedge compression may be started immediately, or a day or two after operation, and within a few days may be maintained almost continuously.

The treatment of extensive and rigid deformities belongs to the province of orthopedic surgery, so will not be discussed in this presentation.

SUMMARY

1. Thoracogenic scolioses are of two types, pleurogenic and thoracoplastogenic. Both are preventable and are curable if corrected in their incipiency.

2. Pleurogenic deformities can be prevented by (1) the prevention of the formation of pleural scar by early cure of the empyema or pleuritis, and (2) the constant attention to maintenance of straight alignment or preferably of overcorrection of the spine. This can be accomplished by means of (a) postural wedging, (b) corrective body casts of plaster of Paris, and (c) resection of ribs on the side of concavity. To facilitate correction and the use of corrective measures, it is helpful to anesthetize the area of the wound by crushing the intercostal nerves which innervate that area.

3. Extensive and rigid deformities of the spine and costal framework are incurable.

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ACUTE EMPYEMA*

The Use of a Bradford Frame to Promote Dependent Drainage and to Prevent Scoliosis

J. Dewey Bisgard, M.D.
Omaha, Neb.

METHODS for prevention and treatment of pleurogenic or empyema scoliosis have been presented in previous publications.[1, 2, 3] As a supplement, a brief report of a more recently used method, utilizing the Bradford frame follows:

A standard frame is suspended upon blocks in the usual manner so that it rests six or more inches above the surface of the bed. The gap

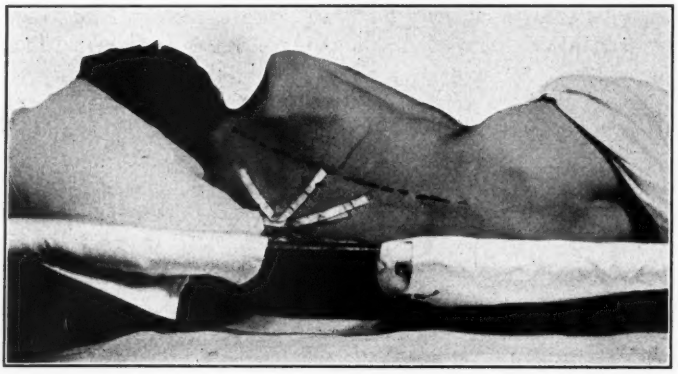

Fig. 1.—A patient with a dependently drained encapsulated interlobar empyema of the left chest, lying upon a Bradford frame upon the affected side which, with the drainage tube, protrudes through the gap free from pressure. In this position drainage is dependent, the spine is bent into a position of overcorrection, and the unaffected side has unrestricted motion.

between the canvas sheets, which ordinarily is placed to accommodate the buttocks, is shifted toward the head end sufficiently to leave the lower half of the chest unsupported. Thus the patient can lie comfortably and almost constantly upon the affected side, with the drainage tube protruding through the gap. With pillows under the head and shoulder, the dependent side will sag into the gap, diverting the spine into position of overcorrection as shown in Fig. 1. Also with the affected side down, drainage may be made more dependent and the diseased side somewhat immobilized by the superincumbent weight, while the normal unaffected half of the chest is given a maximum of unrestricted motion.

*From the Department of Surgery, University of Nebraska School of Medicine.

The heart and other intrathoracic structures gravitate into the affected side, encouraging obliteration of the empyema cavity and increasing the capacity for ventilation of the lung on the unaffected side.

In the case of the patient shown in the illustrations, the use of the frame aided materially in averting a fatal outcome. On the day following drainage of the empyema, there developed signs of pericarditis and heart failure with pulmonary edema. Rapid improvement and recovery followed administration of cardiac stimulants and placement upon the frame with the diseased side dependent.

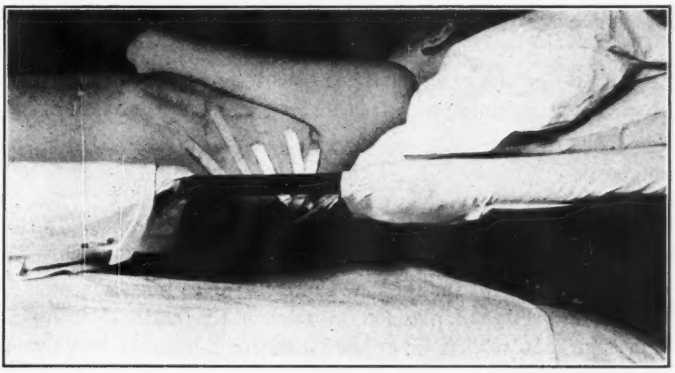

Fig. 2.—Same patient lying upon his back with the operative wound and drainage tube free from recumbent pressure.

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TOTAL PNEUMONECTOMY FOR CONGENITAL CYSTIC DISEASE OF THE LUNG

Report of Successful Case

J. W. Gale, M.D., and J. L. Keeley, M.D., Madison, Wis.;* and H. M. Coon, M.D., Stevens Point, Wis.†

THE reported incidence of congenital cystic disease of the lung indicates that it is a comparatively rare pulmonary disease. The treatment by pneumonectomy of a patient thus afflicted presents the circumstance of an uncommon disease treated by a surgical procedure of rather recent development.

The question of priority in relation to successful total pneumonectomy is somewhat obscured by references to certain early cases. Thus, MacEwen's[1] case was actually the drainage of a large tuberculous cavity surrounded by a thin wall of lung parenchyma, which later sloughed away. Nissen,[2] to whom credit for the first successful total pneumonectomy has been given by Haight,[3] planned to remove the entire left lung for bronchiectasis. In reality, he applied a tourniquet to the bronchus at a secondary operation and exteriorized the lung. Fourteen days later the necrotic lung with some of the silk ligatures about the bronchus came away.

This procedure has also been used by Haight (1932). It differs from the one-stage procedure which has been described by Archibald,[4] Graham and Singer,[5] Overholt[6] and Rienhoff,[7, 8] and which was used in the case reported here, but it still represents total removal of one lung.

We believe the case here reported is the first one of congenital cystic disease of the lung which has been successfully treated by pneumonectomy. The literature on cystic disease of the lung has been reviewed and the condition adequately discussed in articles by Eloesser,[9, 10] Koontz,[11] Miller,[12] Moorman,[13] and Pearson.[14]

CASE REPORT

E. H. E., aged twenty-nine years, was admitted to the Wisconsin General Hospital on March 20, 1934. His chief complaint was coughing up blood. The patient had pertussis at the age of two, since which time he had had a cough. He had been told by his family that the cough was nonproductive in type. In 1919 he had an attack of influenza, manifested by weakness, prostration, fever and cough. One week later this was followed by pneumonia and pleurisy. At that time the cough was productive of rusty sputum. He was in bed four weeks and the condition gradually subsided. Since that time the cough had been productive; the amount was always the

*From the Department of Surgery, University of Wisconsin Medical School.
†From the River Pines Sanatorium.

same, about one cupful in twenty-four hours. He occasionally raised about one cupful of purulent sputum all at once. The sputum was either thick green or yellow in character and had a foul odor. He had never observed whether or not the sputum separated into layers. Since 1919 he has had dyspnea and palpitation on exertion. In 1923, he had an attack of hemoptysis. The amount of blood lost was not known, but it was bright red and he felt faint with the attack. A physical examination of the chest showed no evidence of tuberculosis. In June of 1933, he again coughed up about one cupful of blood, but felt well until March 8, 1934, when he had an attack of frank hemoptysis. This was followed by coughing up of blood for two days, the amount varying from mere streaking of the sputum to about one teaspoonful of pure blood. For about a week, or ten days thereafter, he raised clots with coughing. Since March 8, 1934, he had daily fever ranging from 99.4° to 101° in the afternoon. Since the last attack of hemoptysis, he experienced discomfort in the lower left portion of his chest. Appetite was good and weight had been maintained at 175 pounds. Tonsillectomy had been performed in 1929 with no change in cardiorespiratory symptoms. The sputum had been examined eight times after the hemorrhage in June, 1933, and was found to be negative for tubercle bacilli on each examination. He had experienced no night sweats, had never been exposed to tuberculosis to his knowledge, and had smoked only moderately. Family history revealed no evidence of pulmonary infection.

The pertinent findings on examination revealed the trachea to be deviated to the left. The left anterior portion of the chest was flattened. The left subcostal angle was more acute than the right, and the right clavicle was less prominent than the left. Excursion was less marked on the left than the right. Tactile fremitus was increased over the left chest, and decreased on the right. To percussion there was hyperresonance over the apices, otherwise hyperresonance over the entire right chest. There was dullness to flatness on percussion of the left chest posteriorly. Breath sounds were diminished on the right. There was a loud friction rub in the left upper chest to the fourth rib anteriorly, extending posteriorly to the midaxillary line, high in the left axilla. There were bilateral crepitant apical râles, extending to the level of the fourth rib anteriorly, and to the second, or third spinous process, posteriorly. Amphoric breathing was audible over the fourth interspace inferiorly on the left, and was associated with whispered pectoriloquy; this was also heard in the left axilla, extending posteriorly to the infrascapular line. On the right, amphoric breathing and whispered pectoriloquy were heard in the midaxillary region of the sixth and seventh interspaces. The heart was found to be displaced, the apex lying in the fourth interspace on the left in the midaxillary line; right heart border was found to the left of the midsternal line. There was slight downcurving of the finger nails. Vital capacity was found to be 59 per cent of the expected normal.

Laboratory examinations revealed of interest a total white blood cell count of 13,150 with 83 per cent neutrophiles, 16 per cent small lymphocytes, and 1 per cent monocytes. Sputum examinations were repeatedly negative for tubercle bacilli and fungi, but found to contain streptococci in abundance, pleomorphic gram-negative bacilli, spirochetes and fusiform bacilli. Nose and throat cultures revealed streptococci to be predominating. X-ray of the chest showed the trachea sharply drawn to the left and the heart entirely left-sided, obscured by changes in the left lung field. There was a right scoliosis. The right lung field showed no recent parenchymal change; hilum shadows were obscured by the spine. Basal trunks on the right extended well out to the periphery; the right diaphragm was round and regular; sulcus clear. On the left, no normal architecture could be visualized. There were multilocular rarefactions with thin walls throughout the left lung field, most clearly

visualized along the lateral border from apex to base. The diaphragm was not visualized except by position of gastric air bubble, and appeared to be elevated. Interspaces on the left were narrowed. After a barium meal, the level of the diaphragm on the left was determined by fluoroscopy to be approximately the same as on the right. An attempt to instill lipiodol by the oral route was not entirely satisfactory, but an x-ray following injection revealed pooling of the lipiodol in the left base, a small number of cavities being outlined. The amount of sputum was found to vary between two and seven ounces a day, and became more marked in the presence of an upper respiratory infection. The usual amount, however, was from two to four ounces.

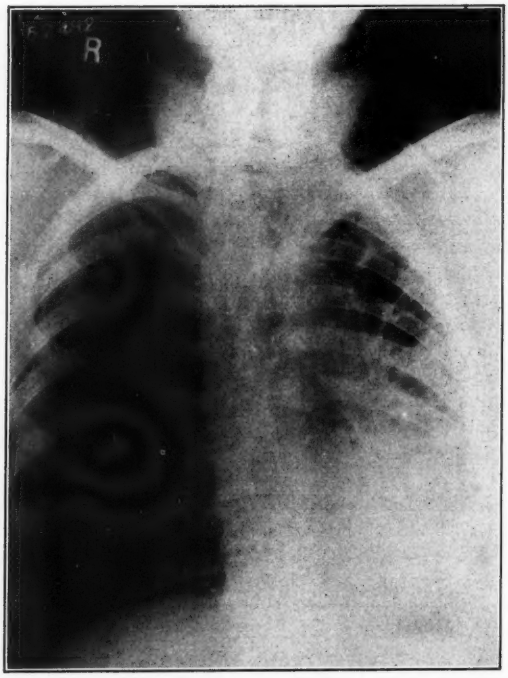

Fig. 1.—This roentgenogram, taken shortly after the patient was admitted to the hospital, shows the right lung field to be clear. The left side demonstrates the multiple cysts in the upper two-thirds of the field. The detail is lacking in the lower portion due to fibrosis and, possibly, fluid. No fluid levels were seen. The trachea is deviated to the left.

He had a mild, intermittent fever. Temperature varied from 98° to 99.4° on the average, with proportionate pulse and respiratory rate. Diagnosis was made of congenital cystic lung disease involving the whole left lung, atelectasis on the left, compensatory emphysema on the right, and displacement of the mediastinum to the left. On April 11, 1934, an artificial pneumothorax was produced on the left side. Roentgenograms of the chest immediately afterward demonstrated a zone of pneumothorax, extending along the left lateral chest wall from the apex to the base, the latter being adherent to the diaphragm. On the following day, an additional quantity (450 c.c.) of air was introduced, leaving pressure readings of -1.5 and -3.5.

On April 16, 1935, a left phrenicectomy was performed which produced a slight elevation of the left half of the diaphragm. Collapse of the cystic lung by thoracoplasty was thought advisable, and the previously instituted artificial pneumothorax was aspirated in two stages.

On May 2, a first-stage paravertebral thoracoplasty was performed with the removal of segments of the first four ribs. The x-ray on the tenth postoperative day showed very good collapse at the apex. A second stage was performed on May 18 with the removal of segments of the fifth, sixth, seventh, and eighth ribs. The collapse of the base was still inadequate and on June 11 a third stage was performed, removing segments of the ninth, tenth, and eleventh ribs. This was followed on July 5 with an anterolateral thoracoplasty and the removal of the remaining

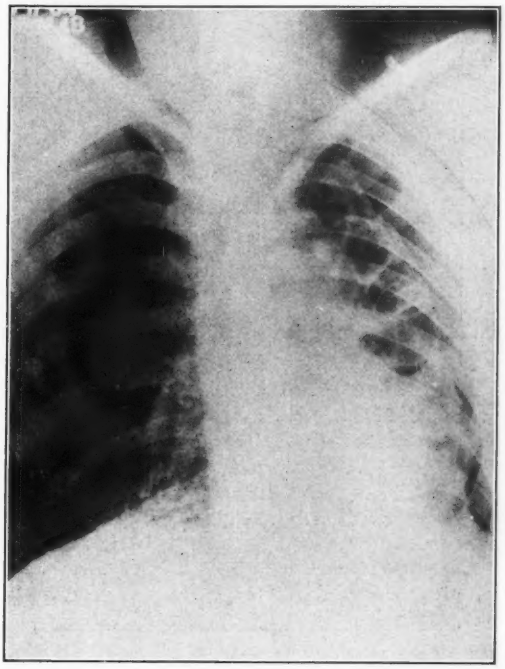

Fig. 2.—Lipiodol has been introduced into the tracheobronchial tree and is shown distributed through the medial basilar trunks on the right. The presence of an artificial pneumothorax on the left, introduced for diagnostic purposes, demonstrates the lateral lung border from the apex to the diaphragm. Adhesions at the base were also determined from this picture. There has been a return of the trachea to the midline, following the increase in intrapleural pressure on the left.

anterior segments of the first, second and third ribs with their respective cartilages. The anterior segment of the fourth rib was removed but the cartilage was allowed to remain to protect the pericardium. X-ray at this time still showed areas of rarefaction at the site of incompletely collapsed cysts. The temperature, however, remained normal and after twenty-four hours the amount of sputum was reduced to considerably less than an ounce, and the patient was gaining weight when discharged from the hospital on July 27. He was seen in the out-patient clinic on August 25, and reported that his expectoration had not decreased since discharge, but that he felt stronger. His only complaint was marked dyspnea on exertion. X-ray taken at

this time revealed essentially the same findings as at discharge. Further x-ray studies in December showed no change, but his dyspnea had improved. On January 4, 1935, he was again operated upon, and the regenerated segments of the resected ribs were removed. This operation was terminated before being completed because of the patient's poor condition. The films on January 17 showed persistent incomplete collapse. On February 5, the removal of the remaining regenerated segments was carried out, and pericostal sutures (15) were used to increase the degree of collapse. X-rays on February 18 showed no lung architecture on the left side, and the collapse was thought to be excellent. A vital capacity determination on February 19 was 20 per cent of normal. The patient was discharged from the hospital on February 21, free from sputum, but very dyspneic. During the next few months his weight, which was 165 pounds at discharge, increased to 180 pounds, and his shortness of

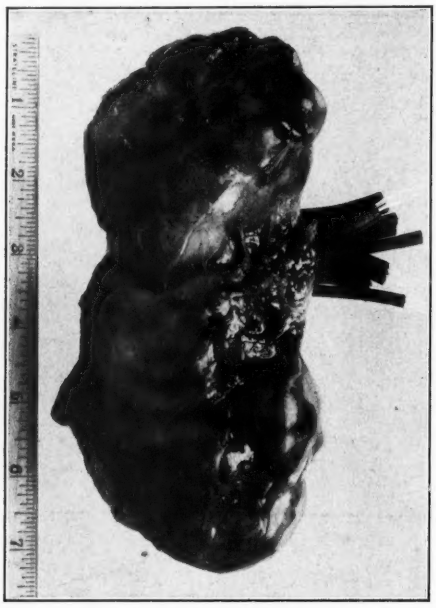

Fig. 3.—The lung has been inflated by means of tubes placed in the bronchial openings. The posterior and posterolateral surfaces are shown. The lobulated appearance is due to the underlying cysts.

breath improved. This improvement did not continue and upon his reentry to the hospital on September 9, 1935, he complained of slight hemoptysis, a persistent cough, loss of ten pounds in weight, and one to two ounces of foul, blood-streaked sputum daily. His dyspnea had again increased subjectively, although his vital capacity was 36 per cent, or 16 per cent, above the level at the time of discharge from the hospital one year previously. On September 25, a total pneumonectomy was performed on the left side. Numerous dense adhesions were encountered but these were divided without undue difficulty. Airtight drainage was instituted postoperatively, and the patient ran an uneventful course except for an acute coryza which he contracted several days after operation. He was discharged from the hospital on December 12, seventy-three days after the operation. His vital capacity was 25 per cent and daily sputum was diminishing, being up to one-half ounce per

day. He was last seen in the out-patient department on January 28, 1936, at which time his vital capacity was 33 per cent; there was a slight gain in weight to 171 pounds; cough had markedly decreased, and there was only occasional sputum.*

DISCUSSION

In dealing with this case, we were confronted with several problems. None of these could be solved through precedent; therefore, it is not strange that we made some mistakes in judgment, as well as in our choice of procedures to be employed. In view of this, it would seem worth while to present our reasons for the method of attack instituted.

When this patient first came under our observation, we were aware that we were not dealing with a simple case of congenital cystic disease

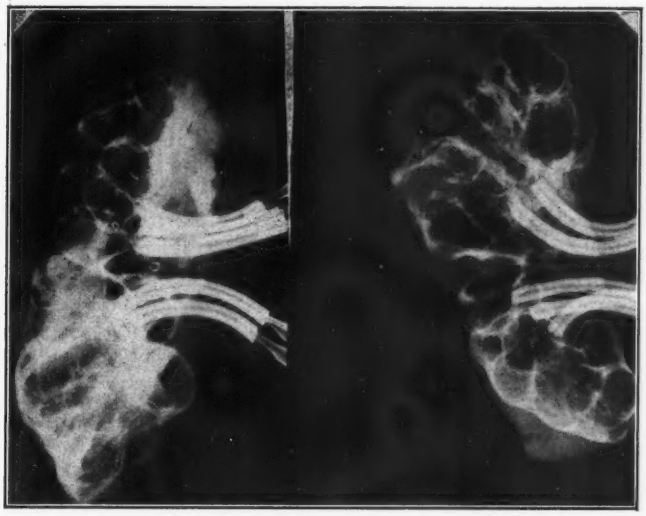

Fig. 4.—Photograph of a roentgenogram of the specimen illustrated in Fig. 3.

of the lung, but also with added complications of an increasing frequent hemoptysis and a superimposed chronic infection. We felt that some form of collapse therapy would be the method of choice. This would be most likely to control the hemoptysis and at the same time reduce the infective process through better drainage.

Artificial pneumothorax was instituted first, because it was the simplest and least dangerous procedure offering a chance of controlling both the hemoptysis and the infection. It was also thought that we could arrive at a better understanding as to whether the infective process had progressed far enough to obliterate the pleural space to such

*Since this manuscript was completed, it has been learned that Dr. J. Dewey Bisgard, of Omaha, performed a total one-stage pneumonectomy for congenital cystic disease of the left lung, July 17, 1934. The patient died twenty-four hours after the operation, presumably from atelectasis of the right lung. (Personal communication.)

an extent as to preclude any chance of pneumonectomy at a later date. It was clear from the first that artificial pneumothorax was not without danger because of the thin cyst walls throughout the periphery of the lung which might easily be torn, resulting in a complicating empyema. The result of the pneumothorax was a partial collapse of the lung, but complicating widespread adhesions about the base caused us to feel that an attempt at pneumonectomy would be technically impossible. This conclusion was later found to be incorrect. Sufficient improvement did not occur following the artificial pneumothorax to warrant its further institution. The lung was then gradually inflated by withdrawing small amounts of air on successive days.

Phrenicectomy was then chosen because, although the lung at the apex had been well collapsed, the symptoms still persisted without improvement. It was felt that the source of hemorrhage and increased density at the base was the site of the real trouble, and that relaxation of the lung base, plus a certain degree of collapse in this region, was indicated. Furthermore, there was no functioning lung tissue on this side, and the permanent loss of diaphragmatic movement could not cause any future disturbance in the patient's vital capacity. It would also enable us to get a better collapse with a thoracoplasty which would most likely be very necessary in the near future. Phrenicectomy did bring about some improvement. The sputum decreased from 150-160 c.c. to 60-80 c.c. daily. There was still, however, some streaking with bright red blood. After one month no further improvement could be seen and a first stage paravertebral thoracoplasty was done. This was followed, in turn, by a second and third stage. Very little improvement was evident and this was attributed to the incompletely collapsed lung. Since the condition persisted, an anterolateral thoracoplasty was done, removing the remaining anterior segments of the first, second, third and fourth ribs, and their respective cartilages except the fourth, which was left remaining over the pericardium. This resulted in temporary improvement again, but the lower portion of the lung had to have further collapse. This was accomplished through the posterior approach in two stages. The x-ray showed excellent collapse and the sputum had almost entirely disappeared at the time of discharge. We felt that we had at last accomplished a sufficient collapse which might be permanently effective. During the following five months, improvement continued; the weight increase was ten pounds, no sputum, and an increase of his vital capacity from 20 to 30 per cent of the normal. He was seen one month later with 30-90 c.c. purulent sputum daily, loss of weight and increasing weakness. He was advised to enter the hospital again for further surgery. We felt that we had accomplished all that was possible through collapse procedures. It was quite evident that complete allevia-

tion of his symptoms could be accomplished only by one of two procedures: (1) cautery drainage, (2) total pneumonectomy. The latter procedure was chosen with the view in mind, that if it were technically impossible to remove completely the entire diseased process, we could always resort to cautery drainage. It was indeed reassuring, when upon opening the thoracic cage for pneumonectomy, we found a lung which had apparently not become more adherent than at the time of the patient's first admission before any operative procedures had been instituted. Following this operation, the vital capacity dropped to 16 per cent, a decrease of 34 per cent, before any collapse therapy was instituted. Since his discharge from the hospital, the patient's weight and strength have increased and the vital capacity has risen to 33 per cent of the normal. Sufficient time (six months) has not elapsed to determine the final outcome of this case, but progress to date is encouraging.

SUMMARY

Experience with one case of infected congenital cystic disease of the lung gives evidence that the treatment should be essentially the same as that instituted for advanced bronchiectasis.

The progress of the disease may be temporarily checked by collapse procedures, but ultimate success can only be obtained through the complete removal of the diseased process as in the reported case. Cautery drainage might well be of value where the infection is limited to a localized area.

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CONGENITAL CYSTIC DISEASE OF THE LUNG*

HARRY G. WOOD, M.D.
ROCHESTER, MINN.

OUR present-day knowledge of, and clinical interest in, cystic disease of the lung has largely developed since the report and review of the literature published by Koontz in 1925. Reports of cases have appeared with increasing frequency, and it is now evident that this condition is not at all uncommon.

Many theories have been advanced to account for it. None, however, is satisfactory and the etiology is still unknown. Some observers still dispute its congenital origin. In some cases, however, infants are found to have cystic disease of the lung, and in all cases in which a pathologic examination of the tissue involved is made, an absence of pigment has been noted. If the condition were not congenital in origin, this should not be so. A varied nomenclature has been employed, but until the real etiology is known the general term "congenital cystic disease of the lung" seems to be the most satisfactory.

In a general way all cases may be divded into two groups: (1) those in which there are single or multiple large cysts containing fluid or air, and (2) those in which there is diffuse degeneration resulting in the so-called honeycomb type of lung. A series of forty-eight cases have been encountered at the clinic in which, I believe, a presumptive diagnosis of congenital cystic disease is warranted (Table I). Sixteen of these cases were reported in 1934; the balance has been collected since. I wish it understood, however, that I am not reporting these as a series of forty-eight positive cases of congenital cystic disease of the lung. I have discussed the question in the Section on Roentgenology at the clinic. and issue is taken with me, on the ground that in some of the cases the roentgenologic findings are not characteristic enough to warrant a positive diagnosis of congenital cystic disease.

SYMPTOMS

While there is no syndrome on which a definite clinical diagnosis can be made, the history is important. Recurring attacks of severe dyspnea with cyanosis, particularly in infants and children, should make one suspect cystic degeneration. In older individuals, a progressive dyspnea, with or without a preceding respiratory infection, warrants the same suspicion.

*From the Division of Medicine, the Mayo Clinic, Rochester, Minnesota.
Read before the Nineteenth Annual Meeting of the American Association for Thoracic Surgery at Rochester, Minnesota, May 4, 5, 6, 1936.

The clinical manifestations vary greatly (Table I). In some cases they are severe and lead to total disability; in others, they may be slight or entirely absent. Their severity depends upon two factors: (1) the extent of destruction of parenchymal tissue, and (2) the change in intrathoracic pressure. An increase in pressure may occur in those cases in which there is a large air-containing cyst with an imperfect bronchial communication. In such cases, sudden attacks of severe dyspnea with cyanosis, closely simulating spontaneous pneumothorax, are prone to occur. Eloesser has stressed the fact that patients with a free bronchial communication, or no communication, do not have these attacks.

TABLE I

CONGENITAL CYSTIC DISEASE OF LUNG (FORTY-EIGHT CASES)

Patients:	
Males	37
Females	11
Ages:	
Youngest, months	15
Oldest, years	79
Lesions wholly or chiefly limited to:	
Right side of thorax	23
Left side of thorax	10
Bilateral	15
Pulmonary symptoms present	38
Other findings:	
Definite tuberculous lesions (all healed)	3
Tuberculous otitis media	1
History of tuberculosis (?)	4
Tertiary syphilis	3
Pulmonary lesions asymptomatic	10
Cysts containing fluid	6
Large air-containing cysts	13
Honeycomb type lesions	29
History of pneumothorax (?)	8
Symptoms:	
Moderate to severe dyspnea	30
Moderate to severe cough	23
Hemorrhage or hemoptysis	6
Severe palpitation	4*
Pain in thorax (only complaint)	4

*In one case associated with cardiac decompensation.

In a number of such cases reported, aspiration of air, or drainage by tube, has been necessary as a lifesaving measure. If increased pressure does not result, aspiration is of no benefit. This was demonstrated in several of the cases in the present series.

DIAGNOSIS

While in some cases the history may suggest cystic degeneration, a positive diagnosis cannot be made on the history and physical examination, the diagnosis in all cases being dependent on the roentgenologic findings. In some instances, bronchoscopic injection of lipiodol before

roentgenography may be necessary; in others, when a balloon cyst is present, induced pneumothorax to force the wall of the cyst away from the thorax may be necessary before the lesion can be differentiated from spontaneous pneumothorax.

In some cases in which cysts contain fluid, but no fluid level is apparent, the lesion may be indistinguishable from a thoracic tumor. In such instances, the diagnosis can be made only by exploratory thoracotomy. A few cases are reported in which a cyst containing fluid has been mistaken for localized empyema, and in these cases the diagnosis was made only when thoracotomy was performed. In general, however, I believe a roentgenologist, who is thoroughly familiar with the condition, can in the majority of cases make a positive diagnosis of congenital cystic disease of the lung on the primary roentgenologic findings. Fluid-containing cysts must be differentiated from: (1) thoracic tumor, (2) pulmonary abscess, (3) dermoid, (4) echinococcus cyst, and (5) empyema.

In cases of diffuse saccular degeneration, the physical findings are unimportant. In some of these cases the roentgenologic findings may suggest bronchiectasis; in most instances, however, they are sufficiently characteristic to warrant a positive diagnosis.

If a large, balloon cyst is present, only one lesion can be mistaken for it, and that is spontaneous pneumothorax. In five of the cases in the present series such a diagnosis had been made. The physical findings are the same for both lesions. As mentioned before, induced pneumothorax may be necessary before the two lesions can be differentiated roentgenologically.

TREATMENT

The treatment at best is unsatisfactory. Some cases are reported in which definite subjective improvement has occurred following bronchoscopic aspiration and injection of lipiodol. One of these forty-eight cases came within this category. I believe, however, that this procedure should be limited to cases of infected cysts that can be successfully aspirated through the bronchoscope.

Some fluid-containing cysts have been successfully removed surgically. Two in the present series were so treated by Dr. Harrington, with apparent cure. In one of our cases, in which a single, large, air-containing cyst was present, there were recurring attacks of dyspnea and cyanosis. In this instance, the attacks subsided without any treatment, presumably as the result of spontaneous closure of an imperfect communication with the bronchus. In another case, in which there was a large, balloon cyst filling the entire right thorax, thoracotomy was performed. The bronchial communications were cauterized, and the patient remained in good health for ten years; death occurred finally as the result of a totally unrelated illness.

For the diffuse saccular, or honeycomb, type of bilateral cystic degeneration no treatment seems worth while.

SUMMARY AND CONCLUSIONS

Congenital cystic degeneration, while not at all common, does occur much more frequently than was previously supposed.

A clinical diagnosis can never be made on the history and physical findings alone. In every case, careful roentgenologic study is necessary. With increased knowledge of the condition on the part of radiologists, satisfactory diagnosis can be made in nearly all cases by careful roentgenologic study.

Secondary pulmonary infection is the greatest danger to which such patients are subject. Patients who have no communication between the cyst and the bronchus are safer than those in whom such a communication exists. Surgical measures to occlude such a communication are a proper procedure, as is also extirpation of fluid-containing cysts.

Patients with infected cysts which can be successfully aspirated through the bronchoscope may benefit by injections of lipiodol. Diffuse bilateral cystic degeneration of the honeycomb type is not benefited by any form of treatment.

A STUDY OF VENOUS PRESSURE AND CIRCULATION TIME IN PULMONARY TUBERCULOSIS*

ALLAN HURST, M.D., AND MORRIS A. BRAND, M.D.
NEW YORK, N. Y.

ALTHOUGH reports of many studies of the circulation time and venous pressure in pulmonary disease appear in the literature, especially that of foreign countries, such studies have not included large groups of pulmonary tuberculosis. These studies have usually had reference to the venous pressure, but there has been no comprehensive study of circulation time in this disease. In view of the complexity of the pathologic processes in pulmonary tuberculosis, it might be expected that there would be changes in the pulmonary circulation in this disease. For this reason, we undertook a study of the circulation time in addition to the venous pressure in all types of pulmonary tuberculosis. In addition, the influence of various forms of collapse of the lung on the pulmonary circulation was studied by these means.

There are several interesting reports in the literature bearing on this subject. For example, in chronic pulmonary emphysema, Tarr, Oppenheimer and Sager[1] found that the circulation time, as measured by the injection of sodium dehydrocholate, was nearly always normal. The exception occurred in their cases of true cardiac decompensation associated with this disease when the time was increased. Weiss and Blumgart,[2] with the radium C technique, found the circulation time and venous pressure normal in this disease, and suggested that these tests be used to distinguish between cardiac and "ventilatory" dyspnea. In a variety of pulmonary conditions, and particularly in silicosis, Miller[3] found no change in the circulation time. Griffith, Chamberlain and Kitchell[4] found no rise in the venous pressure in allergic asthma, pneumonoconiosis, or chronic pulmonary tuberculosis.

In cases of pulmonary tuberculosis with pneumothorax, Hirschsohn and Maendl[5] studied the circulation time by means of the calcium chloride method. These authors showed that the circulation time was within normal limits. Kahler,[6] using the same technique, confirmed these results in a larger series of cases.

With respect to the venous pressure, measurements of this value have been made for a much longer period in both cardiac and pulmonary diseases. In pulmonary tuberculosis, with and without pneumothorax, the French clinicians, notably Villaret[7, 8, 16] and his coworkers, Saint-

*From the Tuberculosis Division of the Montefiore Hospital, Service of Dr. H. Wessler.

Girons,[8] Grellety-Bosviel,[8, 9] Martiny,[10, 16] and Desoille[7] have reported important studies. We shall discuss these later in connection with our results.

This report on circulation time and venous pressure is based on a study of 153 cases of pulmonary tuberculosis in which every type of involvement of the lungs is represented. Seventy-two of these cases were receiving some form of collapse treatment, and in some of them a thoracoplastic operation had been performed.

TECHNIQUE

The method chosen for the determination of circulation time was the arm-to-tongue time interval measurement following the injection of saccharine. It was not deemed necessary to study the pulmonary circulation time by means of the ether method, because it was found, after injection of saccharine in a small number of cases, that the total circulation was within normal limits.

The method for venous pressure determination was as follows: The patient was placed in the horizontal recumbent position with the arm resting on a pillow. The level of the antecubital vein was estimated as 4 or 5 cm. below the level of the right auricle. A long tube was calibrated into centimeters and bent at one end to fit into an 18 gauge needle. The tube was first flushed with 3 per cent sodium citrate and was inserted into the needle after the syringe, with which the saccharine was injected for the circulation time determination, was detached. The level of blood within the tube was taken as the venous pressure reading. To determine whether clotting of the blood had occurred in the vein, tube, or needle, pressure was applied proximal to the venipuncture, meanwhile, observing the rise and fall of the column of blood in the tube.

The direct cannulation of the vein has been criticized as a method of studying the venous pressure. The use of a soft object, such as a pillow, introduces an error, since the level of the vein below the right auricle is a factor in determining the variation in venous pressure when measured at intervals. Correct posture and body rest prior to testing were always insisted upon. Attempts at rigorous accuracy were made by repeating the observations in those cases in which abnormal readings were obtained.

The normal venous pressure with this method was found to be 5 to 9 cm., although some authors give the maximum reading as 15 cm. Villaret uses a high normal of 13 cm. for men, and 12 cm. for women. We have taken the latter figures as our high standard.

RESULTS

We shall first summarize our observations with respect to the circulation time as follows: In 153 cases of pulmonary tuberculosis, which include those receiving collapse therapy and those without any form of

collapse, the average circulation time was 12.73 seconds. A further analysis of these cases shows that in the 81 cases without collapse therapy, the average circulation time was 12.7 seconds.

In the entire series of cases, the circulation time was within normal limits (12-16 seconds), except in one case, in which the readings were 19 and 20 seconds, respectively, on two determinations. The patient was an emaciated, diabetic female with extensive bilateral tuberculosis, and it was felt that with our method of testing for circulation time, a large subjective element was present; because of the clouded sensorium of this patient, the results were not trustworthy.

We shall devote the rest of the analysis to a presentation of our findings with respect to the venous pressure. This was usually determined only in the arm on the side of the pulmonary lesion, or on the same side of the lung receiving collapse therapy. In most cases, if the venous pressure reading was a high normal, or above normal, a determination was made in both arms. In those cases where the test was performed on both sides, it is so indicated.

In 81 cases of pulmonary tuberculosis chosen at random and representing every type of involvement, but without collapse therapy, 75 cases had a venous pressure within normal limits. The average was 7.9 cm. The cases with abnormal readings were as follows:

	Venous Pressure	
	Right	*Left*
1. Extensive fibrocaseous tuberculosis with slight retraction to the right.	17 cm.	--
2. Bilateral extensive tuberculosis with slight retraction to the right. Patient was very dyspneic. No signs of cardiac failure.	16.5 cm.	16.5 cm.
3. Bilateral upper lobe extensive tuberculosis. Slight dyspnea. No signs of cardiac failure.	15 cm.	18 cm.
4. Severe cirrhotic, contracted atelectatic left lung with emphysema of the right lung.	12 cm.	13.5 cm.
5. Extensive caseocavernous tuberculosis of the right lung with retraction of the heart and mediastinum to the right.	23 cm.	3.5 cm.
6. Extensive fibrocaseous tuberculosis of the right lung with marked retraction of the heart to the right. Dyspnea on exertion.	22 cm.	5.5 cm.

In the group of 72 cases of pulmonary tuberculosis with collapse therapy, there were 19 cases of thoracoplasty and 53 cases of pneumothorax.

The thoracoplasty cases: Twelve cases without any preoperative figures for comparison were studied after the completion of one or more stages. The venous pressure was normal in 11 of these, but in the twelfth case (this patient had undergone a three-stage thoracoplasty with two revision operations on the left), there was a rise to 13.5 cm. on the left side, and 14.5 cm. on the right. In the 7 remaining cases, studies were made both pre- and postoperatively, and of these, three patients showed

no appreciable change in venous pressure. The 4 cases in this latter group are listed with slightly more detail:

	Venous Pressure	
	Preoperatively	*Postoperatively*
7. Cavity in the left upper lobe.	(L) 11 cm.	(L) 6.5 cm. (second stage)
8. Cavity in the left upper lobe.	(L) 12.7 cm.	(L) 6 cm. (second stage)
9. Cavity in the right upper lobe.	(R) 7 cm.	(R) 15.5 cm. (second stage) (R) 9.5 cm. (2 weeks later) (L) 6.5 cm. (2 weeks later)
10. Cavity in the right upper lobe. Death due to spread of disease.	(R) 13.5 cm.	18.5 cm. (1 week later)

The pneumothorax cases: Of the 53 cases of pneumothorax studied, venous pressure studies were made in 32 cases only after the induction and only for a relatively short period afterward, with little or no change in the degree of collapse; 30 of the 32 cases had either unilateral or bilateral pneumothoraces with various degrees of collapse. Several had either moderate or massive effusions, with or without mediastinal deviation. The other two were cases of inhibition oleothorax. In 30 of the entire 32 cases, the venous pressure was normal. The two cases with abnormal findings were as follows:

11. With bilateral pneumothorax.
    Right venous pressure: 20 cm. A small encapsulated pneumothorax at the apex under high tension.
    Left venous pressure: 7 cm. Selective collapse under negative pressure.
12. Bilateral pulmonary tuberculosis with pyopneumothorax on the left and mediastinal shift to the right.
    Right venous pressure: 7 cm.
    Left venous pressure: 21.8 cm.

In the 21 cases of pneumothorax followed, either before and after the induction of collapse, or during the course of collapse, the figures are much more interesting. While the venous pressure in many cases remains within normal limits, the variations during the course of collapse therapy may be of aid in demonstrating the mechanics involved. The changes, however small, when found consistently on repeated examinations, are of definite significance. There were 10 cases in this group, all without mediastinal shift. Among this latter group there was one case of bilateral pneumothorax, and one case with a moderate effusion. The venous pressure in the 10 cases remained stationary. The remaining 11 cases will be given in slightly greater detail because of the interest involved.

	Venous Pressure	
	Right	*Left*
13. Atelectatic, retracted tuberculosis of the left lung with shift of the heart to that side.	--	14.5 cm.
After pneumothorax and left phrenicectomy.	--	7 cm.
14. Pneumothorax on the right with large cavity in the right upper lobe (low tension pneumothorax).	6.5 cm.	--
During high tension pneumothorax.	11.6 cm.	--
After relaxation of pneumothorax.	6.5 cm.	

	Venous Pressure	
	Right	*Left*
15. Tuberculosis of right lower lobe (before pneumothorax).	6.2 cm.	
Postpneumothorax with slight deviation of the mediastinum.	13 cm.	--
16. Tuberculosis of the left upper lobe (before pneumothorax).		6 cm.
Pneumothorax with slight shift.		11.5 cm.
17. Extensive fibrocaseous tuberculosis of the left upper lobe with retraction of the heart and trachea to the left.	--	3.5 cm.
Pneumothorax with shift to right.	--	14.5 cm.
Pneumothorax with less shift.	--	12.5 cm.
18. Right pneumothorax with selective collapse of the right upper lobe and a fixed mediastinum. High tension pneumothorax.	19.5 cm.	--
Relaxation with negative pressures.	12.5 cm.	--
19. Small bilateral pneumothoraces with apical adhesions on both sides.	9 cm.	--
With increased pressure.	17.4 cm.	11 cm.
20. Extensive tuberculosis of the right lung.	8.5 cm.	--
After induction of pneumothorax. Poor collapse because of adhesions. Shift of the mediastinum to the left.	9.5 cm.	23.1 cm. (paradoxic)
21. Pneumothorax on the right with 50 per cent collapse. Lung adherent to apex and diaphragm.	13 cm.	--
After right phrenicectomy and high tension pneumothorax.	6 cm.	--
22. Pneumothorax on right with 50 per cent collapse. Adhesion to apex. No mediastinal shift.	4 cm.	--
Increased collapse with effusion.	11.5 cm.	--
23. Fibrocaseous tuberculosis, left, with retraction.	4 cm.	--
Left pneumothorax with slight mediastinal shift to right.	10.3 cm.	--

DISCUSSION

In this study it was found that in cases of pulmonary tuberculosis, regardless of the extent of the disease and the type and degree of collapse, the circulation time remained within normal limits. This confirms the work of other investigators and emphasizes the statement of Weiss and Blumgart[2] to the effect that the circulation time can be used to differentiate between cardiac and "ventilatory" dyspnea.

In regard to the venous pressure, before entering into a discussion of our results, it would be best first to consider some of the anatomic and physiologic details. According to Best and Taylor,[11] the factors which influence the venous flow are as follows:

1. Contraction of the left ventricle.

2. Quantity of blood flowing through the arterioles from the arteries, in relation to the capacity of the capillaries and veins.

3. Subatmospheric pressure within the thorax.

4. Action of the right side of the heart.

5. Massaging effect of the skeletal muscles, and support afforded by the abdominal wall.

6. Effect of gravity.

The intrathoracic pressure has an important influence upon the flow of blood along the great veins of the thorax and abdomen, and thus upon the filling of the right auricle. During inspiration the pressure within the thorax is about –81 mm. H_2O, while in expiration it is –34 mm. H_2O. Subatmospheric pressure expands the thin-walled intrathoracic veins, and venous blood is sucked into the thorax.

The blood in the great veins at their entrance into the right auricle has a small but definite pressure of 5 mm. H_2O. The "effective venous pressure" is thus the sum of the subatmospheric pressure in the thorax during any phase of the respiratory cycle, plus the small positive pressure in the great veins. The descent of the diaphragm during the respiratory phase also, by compressing the abdominal contents, increases the pressure in the inferior vena cava and augments the flow of blood to the heart. The venous pressure as measured in the peripheral veins of the upper extremity, more especially the median basilic, is 5 to 9 cm., with 11 cm. as the upper high standard. This is the figure taken by Eyster[12] and most of the other American investigators, but the French quote figures as high as 15 cm. Villaret,[7, 8, 16] with his extensive experience, uses a high standard of 13 cm. for men, and 12 cm. for women.

In the normal man, the venous pressure in the larger superficial veins is quite constant in the same individual from time to time. Under conditions of quiet breathing, it has been found that, so far as pressure in the peripheral veins is concerned, there is little variation between the phases of inspiration and expiration. This is due to absorption of the differences encountered in the thorax by inertia of the blood column between the central and peripheral veins (Waud[13]). Definite respiratory variations in pressure in a peripheral vein in quiet breathing are thus absent or small. In the extreme variations in intrathoracic pressure produced during the Valsalva experiment (expiratory effort with the chest in the inspiratory position, and the glottis closed), and during the Mueller experiment (inspiratory effort with the chest in the expiratory position, and the glottis closed), extreme changes of peripheral venous pressure may occur. Pressures in the Valsalva experiment have been recorded as high as 40 to 50 cm. due to the prevention of entrance of blood into the thorax, because of positive pressure in this cavity; in the Mueller experiment, the peripheral venous pressure appears to fall, due to the extremely low intrathoracic pressure.

Obstruction locally of a peripheral vein will, of course, cause a local rise in pressure. Compression of the large intrathoracic veins (cavae) or the right auricle will be followed by a generalized rise in venous pressure. The innominate and subclavian veins occupy a relatively intimate relationship to the lungs, so that physical changes in either hemithorax might cause distortion or compression of the veins with unilateral rise in venous pressure.

In the 81 cases of pulmonary tuberculosis studied without collapse therapy, 75, or 92 per cent, showed a venous pressure within normal limits with an average of 7.9 cm. The majority of these cases had a moderately or far-advanced tuberculosis, and we are thus unable to substantiate the claims of Martiny[10] that this type of case usually shows a low venous pressure. In 6 cases, the venous pressure was definitely raised, and since in 5, the pressures were recorded in both arms, only these will be discussed. In 3 cases (2, 3, 4), the venous pressures were raised bilaterally, practically to similar levels. The differences in the readings in both arms in two cases (3, 4), in spite of repetition, are so small as to be within the margin of error. It is conceivable that the rise is due to twisting of the heart on its vascular base, with resultant torsion of the great veins. There was no clinical evidence of cardiac failure. In two cases (5, 6) with unilateral atelectasis, fibrosis, and retraction, there was an extreme unilateral rise in venous pressure on the homolateral side. In this type of case, there is most likely a torsion of the subclavian or innominate veins. Here thoracoplasty causes its most spectacular change in venous pressure by releasing the local obstruction.

In regard to thoracoplasty, studies by Coryllos and Stanford,[14] and Overholt and Pilcher[15] have shown that the homolateral venous pressure may be raised or lowered by the operative procedure. In our series, there were 19 cases of thoracoplasty, 12 of which were not studied preoperatively. In 11 of these, the pressures after the operation were within normal limits. In the twelfth case, the patient had undergone a three-stage thoracoplasty on the left, with two revision operations. Her pressures were raised bilaterally to 13.5 cm. on the right, and 14.5 cm. on the left. While there was no evidence of cardiac failure, dyspnea on slight exertion was constant. We are not prepared to give a satisfactory explanation for this change without preoperative readings. In Cases 6 and 7, the pressures dropped, and in Cases 8 and 9 they were raised. In Case 8, the pressure on the right rose from 7 cm. to 15.5 cm. after the second stage, and dropped to 9.5 cm. two weeks later. Coryllos and Stanford[14] investigated the venous pressures in 34 patients preoperatively and postoperatively. In 9 cases, the venous pressures either did not change, or became lower. In 21 cases, there was a rise on the side of the operation, and in 4 cases, there was a bilateral elevation more marked on the side of the operation. They point out with justification that a unilateral rise is most likely due to the dropping of the axillary vein with local obstruction, and is, therefore, of no prognostic significance.

Overholt and Pilcher[15] offer a poor prognosis where the venous pressure rises postoperatively, and wait until the venous pressure falls to normal before proceeding with the operation. We are unable to see any relation between a unilateral rise in venous pressure and prognosis as to life.

In pneumothorax with its varying intrapleural pressures, mediastinal deviations, and complicating effusions, one finds the most interesting figures in venous pressure studies. In a total of 53 cases, 32 were studied after a pneumothorax had been induced, and except for repetition of abnormal figures, were not followed. In 30, with all degrees of collapse, and many with effusions and mediastinal deviations, the venous pressures were normal. In 2 cases (11, 12) there was a marked unilateral rise, undoubtedly due to local compression of the subclavian or innominate veins.

In the 21 remaining cases studied either before and after induction of the pneumothorax, or during the course of some intrathoracic physical change, 10 showed, for all practical purposes, no change in venous pressure. These cases had no mediastinal deviation. In the other 11 cases, as pointed out previously, the venous pressure changes are of extreme interest. This is due chiefly to the fact that, although many of the readings remained within normal limits, they showed sufficient variation to demonstrate the mechanism involved. Unfortunately, the great majority of these cases was not studied with bilateral readings. Nevertheless, it is felt that in view of our previous data, these cases may be cited with benefit. Lengthy discussion is unnecessary, and only main features will be given.

Atelectatic retracted lungs with high venous pressure on that side may show, after pneumothorax or phrenicectomy, a reduction in venous pressure by the release in pull on the venous return in the subclavian or innominate veins. High tension pneumothorax may cause a rise in venous pressure on that side, with or without marked mediastinal deviation. Effusions act in a similar fashion by causing a rise in the intrapleural pressure. There is no direct relationship between the shift of the mediastinum and the rise in venous pressure.

It has been shown previously that in the vast majority of cases, venous pressure changes occur on the side of the more extensive tuberculous lesion or collapse measure. This can only be due to local obstruction of the subclavian or innominate veins, and not to pressure on the superior vena cava or the right auricle. The French investigators, Villaret, Cordier, Grellety-Bosviel, Martiny, and others, in all their publications, constantly refer to high venous pressures as due to pressure upon the vena cava or the right auricle. Grellety-Bosviel,[9] in a large series of cases with unilateral pneumothorax, found the venous pressure unchanged in 85 per cent. In the remaining 15 per cent, there was a temporary rise in the venous pressure. This, he believed, was due to a diminution of lung substance and compression of the pulmonary vessels with stasis in the right chambers of the heart.

In a discussion of mediastinal syndromes studied with venous pressure, Villaret and Martiny[16] quote Cordier as saying that in pleurisy with effusion, only those of the right hemithorax cause a rise in venous

pressure by direct pressure on the superior vena cava. They disagree with Cordier and believe that the left may be similarly affected when there is displacement of the heart to the right. "At the beginning of an effusion, the venous pressure remains the same. If the mediastinum is easily mobile in larger effusions on the right, the venous pressure remains normal; however, on the left, with displacement to the right, there is a definite rise in venous pressure. These facts are logical if correlated with the anatomy, since movement of the heart to the right is unnatural." The constant reference to a central compressing mechanism (cavae or right auricle) suggests the failure to make venous pressure determinations bilaterally, with the resultant error. Again they say, "It is important to measure the venous pressure rather than the intrapleural pressure to find the tolerance of the mediastinal structures." A bilateral rise in venous pressure in the course of collapse therapy is so infrequent that, from the clinical point of view, it is unimportant to consider pressure upon the mediastinal structures as a factor in its production.

If venous pressures are determined bilaterally, it can be shown that variations occur almost in every instance on the side of the greatest physical change, whether due to disease or to collapse therapy. These changes are due to direct compression or torsion of the subclavian or innominate veins. They should be viewed only with anatomic interest and have no relation to the prognosis. One should not attempt to draw conclusions from venous pressure changes in one particular case to an entire similar group. There are many factors to be considered in a unilateral rise in venous pressure, notably the lability of the mediastinum, the intrapleural pressure, and more particularly, the speed with which that pressure changes. The last is borne out by observations on spontaneous pneumothorax with and without effusions where the venous pressure rose suddenly on the side involved, to fall again in a few days with absorption of air and lowering of the intrapleural pressure. Again, the fortuitous arrangement of small pockets of air and fluid may cause direct compression of the veins in one case and not in another, with a larger hydropneumothorax.

SUMMARY

1. A study was made of 153 cases of pulmonary tuberculosis with and without collapse therapy, with circulation time and venous pressure.

2. The circulation time (saccharine method) was always normal, the average reading being 12.73 seconds.

3. The venous pressure usually shows variations on the side of the greatest tuberculous change or collapse measure.

4. This change is due to direct compression or torsion of the subclavian or innominate veins, and not to pressure on the right auricle or superior vena cava.

5. In a very small group of cases, the venous pressure is raised bilaterally. It is conceivable that these changes are due to a twisting of the heart on its vascular base, with torsion of the great veins.

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REPORT OF A LARGE THYMIC TUMOR SUCCESSFULLY REMOVED BY OPERATION*

WILLIAM DEWITT ANDRUS, M.D., AND NATHAN CHANDLER FOOT, M.D.
NEW YORK, N. Y.

THE case we are reporting presents a number of interesting features: it is the largest thymic tumor we can find recorded in the literature; it was successfully removed by operative procedure, again a rarity, and, although it is of the unusual nonmalignant variety, it was unaccompanied by myasthenia gravis.

CASE REPORT

History.—The patient, a thirteen-year-old schoolboy, was admitted to the New York Hospital on October 14, 1936, suffering from weakness, dyspnea and orthopnea of seven months' duration. He had been well up to the age of seven, when he had had scarlet fever and, following this, measles and chickenpox. Since that time he had been somewhat frail, had lost considerable time from school, and had been backward in his schoolwork. At the age of nine, he was fluoroscoped by his family physician who told his parents that he had "thymus gland trouble" and prescribed some oral medication.

During the past few years, the boy became easily fatigued and showed definite shortness of breath on exertion. He had only a slight cough without expectoration or hemoptysis. During some seven months prior to admission, these symptoms became aggravated, particularly the dyspnea and orthopnea. The latter was most marked at night and required his sleeping propped up on four or five pillows. He had no chest pain, and his appetite had been good throughout his illness. No gastrointestinal symptoms had been noted and there were none referable to the genitourinary tract, except for enuresis of long standing.

Physical Examination.—Examination showed a fairly well-developed, but frail and chronically ill boy, with moderate dyspnea on lying flat in bed. There was no clubbing of the fingers or toes. The physical findings of note were limited to the chest and neck.

The trachea was considerably deviated to the right, the thyroid was not palpably enlarged. The chest appeared somewhat emaciated, and there was definite fullness in the left hemithorax, where movement was markedly limited on respiration.

The cardiac and superior mediastinal dullness were found to be noticeably displaced toward the right, to the extent of 8 cm. beyond the midline in the second interspace and to 12 cm. in the sixth. The left border could not be made out. The heart sounds were best heard in the fourth and fifth interspaces to the right of the sternum. No murmurs were audible.

The left lung field was obliterated by flatness on percussion below the first rib anteriorly, and below the spine of the scapula posteriorly. Along the spine the note was impaired, but it was not as flat as laterally. Over the area of flatness breath sounds were absent, while in the axilla distant tubular breathing could be heard. The breath sounds were harsh over the right lung field. The diaphragm was evidently lower than normal.

*From the Department of Surgery, Cornell University Medical College, and the New York Hospital, New York, N. Y.

Laboratory Findings.—The urine was negative. Blood: erythrocytes, 5,500,000; leucocytes 16,000. The Wassermann and Klein tests were negative, as was the Mantoux. Stools were also negative. Ten cubic centimeters of chest fluid, obtained by thoracentesis, showed a cell count of 3,300, of which 12 per cent were polymorphonuclears and 88 per cent lymphocytes; it was sterile on culture. Roentgenograms showed a mass occupying the entire left hemithorax except at the extreme apex and base, dislocating the heart and mediastinal structures to the right. The central portion of this shadow was denser than the peripheral. Two aspiration biopsies failed to yield adequate tissue for study, although a few large cells and lymphocytes, suggesting thymic tissue, were noted.

Diagnosis.—The great size of the tumor and the fact that, despite this, signs of mediastinal compression had developed only very recently, led us to feel that we must be dealing with a slow-growing and soft tumor that had adapted itself well to the conformation of the mediastinal structures. This consideration, when added to the x-ray finding of a shadow more dense centrally than peripherally, strongly suggested the preliminary diagnosis of intrathoracic lipoma, and it was with this diagnosis that operation was undertaken on November 6, 1936.

Operation.—With the patient under intratracheally administered ethylene and ether, an incision was made along the sixth rib, extending from the left border of the sternum to the posterior axillary line, and this rib, together with its costal cartilage, was resected. The pleural cavity was then opened by incising the posterior periosteum and parietal pleura, exposing a yellowish mass covered by a glistening membrane. It was of soft consistency and appeared to be lobulated. A gross diagnosis of lipoma was made by both operator and pathologist.

Since more exposure was obviously needed, it was secured by cutting across the costal cartilages of the third, fourth, fifth, and seventh ribs, and retracting them upward and downward. It was then possible to demonstrate that the tumor was covered by the mediastinal pleura and, after incision of this, its removal from within its capsule was begun, clamping and ligating its blood supply as the vessels appeared. The tumor could be peeled out of its capsule much as one enucleates a subcutaneous lipoma. On continuing the dissection along the anterior border of the tumor, it was found to extend across the midline anterior to the parietal pleura on the right as far as the anterior axillary line. After this portion of the tumor had been delivered into the wound, the dissection was carried posteriorly and into the superior mediastinum, where lappetlike prolongations of the growth were found to have insinuated themselves between and around the great vessels.

The last portion of the tumor to be exposed was a tonguelike process, 6 cm. in length, about 5 cm. in width, and 4 cm. in thickness, which lay high in the superior mediastinum between the innominate and carotid vessels. Because the patient was beginning to show the effects of the procedure and because of the hazard of the dissection at this point, the tonguelike piece was left in situ after being amputated from the main body of the tumor.

After the removal of the growth, the left thoracic cavity was entirely empty, except for the left lung, which appeared to be completely atelectatic and occupied the posteromesial and superior portion of the thorax.

Closure of the chest was accomplished by placing three sutures of silver wire about the fifth and seventh ribs and pulling these together. The periosteum of the resected sixth rib was then approximated, the muscles sutured and the skin closed with interrupted sutures of silk. Closed-tube drainage was inserted into the chest through the ninth interspace in the posterior axillary line.

During the course of the operation, which lasted somewhat over two hours, the patient received an infusion of 500 c.c. of normal salt solution as well as a transfusion of 700 c.c. of citrated blood.

The boy stood the operation very well, but because of some dyspnea, he was placed in an oxygen tent for about thirty-six hours. The tube drained about 400 c.c. of serosanguineous fluid during the first forty-eight hours and was removed on the fourth day after operation. The left lung expanded gradually and at the time of discharge from the hospital, twenty-seven days after the removal of the tumor, had expanded to fill the left thoracic cavity completely, as evidenced by x-ray and physical examination.

Pathologic Report.

Gross Examination.—The tumor was a large, yellowish white, lobulated growth, weighing 2,235 gm. and measuring 26 cm. in width, 24 cm. in vertical diameter, and 7.5 cm. in thickness. It showed definite lappets (Figs. 1, 2), the largest, extending

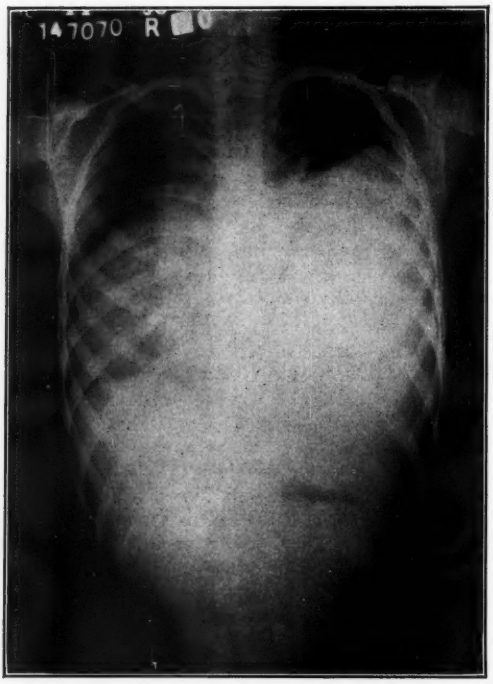

Fig. 1.—Preoperative x-ray showing portion of the chest occupied by the tumor, with displacement of the heart and mediastinal structures to the right, and depression of the left side of the diaphragm. Note that the tumor shadow is less dense at its periphery.

to the right side of the chest, measured 13 cm. in vertical diameter, 14 cm. in width, and approximately 6 cm. in thickness. It differed in no way from the main mass of the neoplasm. The whole tumor was well-encapsulated and smooth on its surface, with a small piece of mediastinal pleura adhering to one margin. Its vascularity was considerable. At one point was a raw area where the tumor had been severed from the lobule that had, perforce, been left in place at the operation. This was at the apex of the growth. Desiring to preserve this huge "lipoma" for museum purposes, it was not completely transsected, small blocks merely being removed at strategic points for microscopic examination. The section surface of these appeared like ordinary adipose tissue, with areas that were dull yellowish white, as though

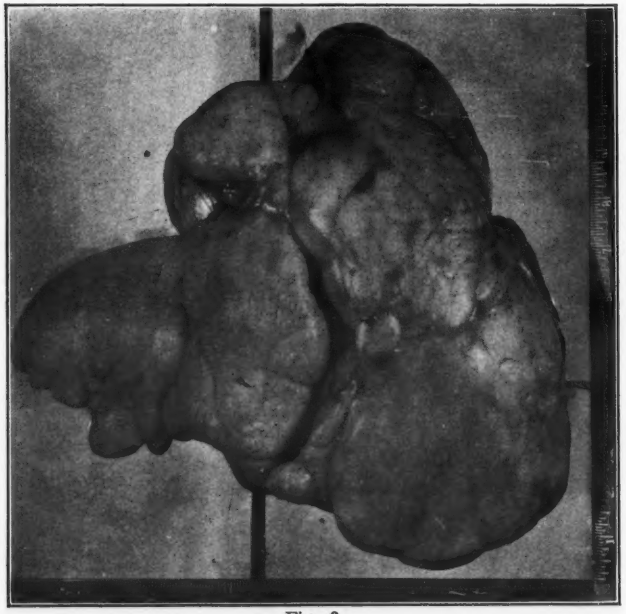

Fig. 2.

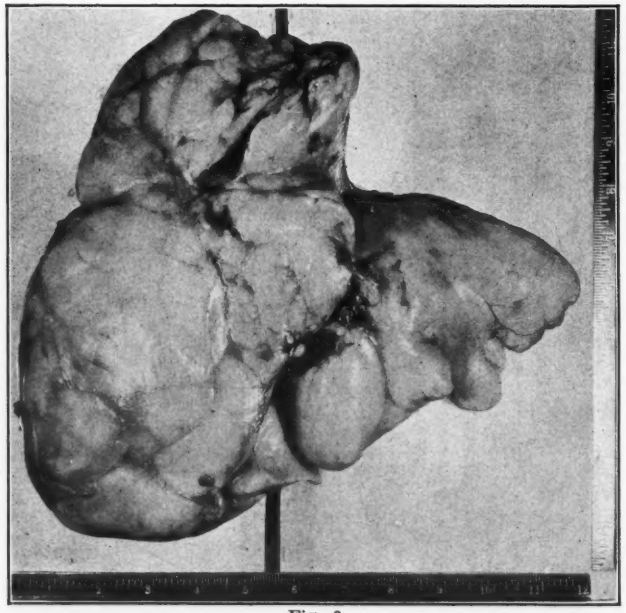

Fig. 3.

Fig. 2.—Gross photograph of the specimen with foot-rules placed near. The dark strip in the background represents the midline of the body, showing the extent of growth to the right.

Fig. 3.—Rear view of the tumor. At the apex is a pedicle by which it was attached to the lappet that involved the great vessels.

fibrosed or degenerated. In retrospect, one may realize that it resembled thymic tissue rich in fatty elements, but at the time of examination the diagnosis of lipoma went uncontested.

Microscopic Examination.—Sections were stained with Masson's trichrome light green (our routine), with hematoxylin-eosin (for photomicrography), with Giemsa's stain (for lymphoid elements), and they were impregnated with silver. Fig. 3 shows the appearance of a typical section, the thymic characteristics being at once manifest. This tissue approached normal thymus so far as its constituents were concerned, but the architecture of the organ was badly disarranged. Blocks were taken at five or six widely separated points in the growth and all showed the same picture. There

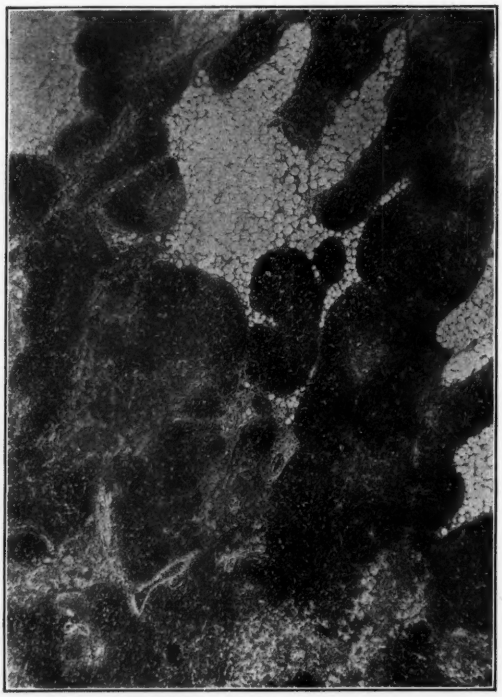

Fig. 4.—Low-power general view of the tumor, to demonstrate its lobular arrangement and the large amount of fatty tissue between lobules. (Giemsa. ×25.)

were small "thymocytes" in abundance, thymic reticulum cells, thymic corpuscles in every stage of development and involution, and a few of the large cells with coarse eosinophile granules that are found in the normal thymus. The reticulum cells were, however, concentrated at the periphery of the lobules, instead of lying at their center (Fig. 4), while the thymocytes lay centrally and did not form the peripheral scallops normally seen. In addition, long cords of epithelioid cells were often noted, sometimes in double rows resembling ducts. The connective tissue showed much hyalin degeneration, often taking on an almost osteoid appearance. Fig. 5 shows the arrangement of the center of the lobules and the frequently noted calcified cores of the thymic corpuscles. The tumor was composed of islands of such "thymoid" tissue embedded in an adipose stroma, some of them quite well

organized, others more primitive. A very few were composed entirely of small cells resembling lymphocytes and actively growing, as indicated by the many mitoses present (Fig. 6). This was the only indication of malignant change in the growth, and it constituted but an infinitesimal part of the tumor. The special stains brought out very little additional data. Silver demonstrated the usual coarse thymic reticulum that was concentrated about the thymic lobules without tending to encroach within their confines (Fig. 7). Giemsa's stain intensified the resemblance of the thymocytes to rather immature lymphocytes.

Diagnosis.—Essentially nonmalignant thymoma with rare anaplastic areas.

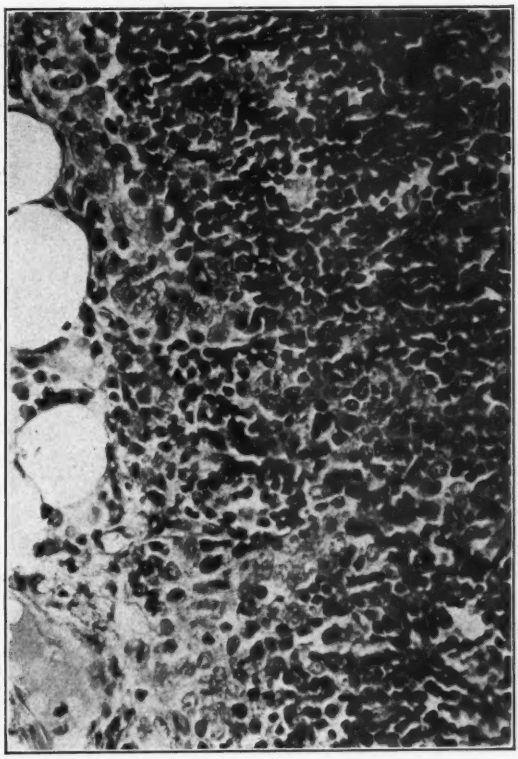

Fig. 5.—Higher magnification to show margin of a lobule with peripheral crowding of thymic reticulum cells. (H. and E. ×800.)

DISCUSSION OF THE PATHOLOGIC FEATURES

The picture presented was much the same as that described by several authors in their reports on thymomas, all fairly recent. Danisch and Nedelmann[1] present a very similar tumor that gave rise to widespread metastases, notably in the nerve roots of the medulla and cord, even involving the cauda equina. Alter and Osnato[2] report a nodule composed of thymocytes and thymic reticulum cells, but containing no Hassall's corpuscles. It was arranged in the lobulated fashion of

thymus and accompanied by numerous "thymic granulomas" and generalized lymphoid hyperplasia. Symptoms of myasthenia gravis were present, and they consider this to be a form of status thymicolymphaticus. Goyle, Vasudevan and Krishnaswamy[3] report a tumor composed of small cells resembling thymocytes and occasional groups of larger ones, resembling thymic reticulocytes, sometimes differentiated into rudimentary thymic corpuscles. We had the privilege of examin-

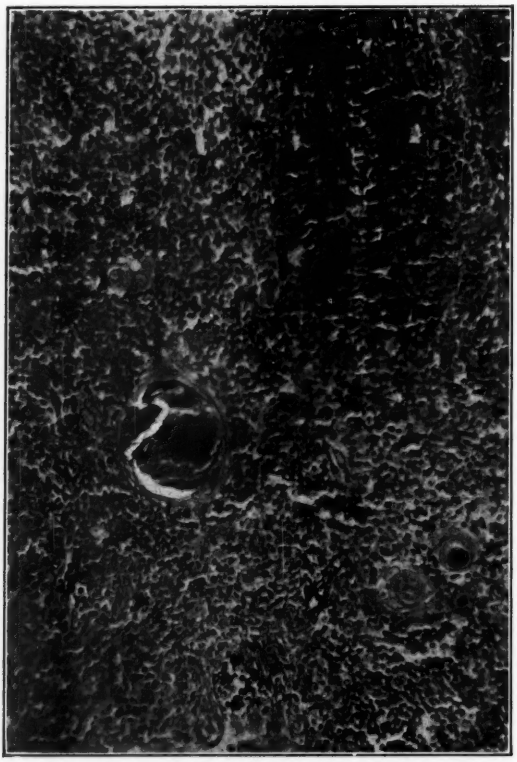

Fig. 6.—Thymic corpuscles in the tumor, the reticular framework and follicular arrangement of "thymocytes" is seen. The dark areas in Hassall's corpuscles are lime salts stained with hematoxylin. (H. and E. ×200.)

ing slides from their tumor and submitting them to Dr. Ewing, who agreed as to their representing an unusual form of thymoma. Gold,[4] Alajouanine and others,[5] and Norris[6] have all described similar tumors of a predominatingly small-celled, usually nonmalignant type.

It is evident, then, that the tumor we are reporting belongs to the rarer group of nonmalignant thymomas and is one that substantiates the validity of calling such neoplasms by the name of the organ in which they occur. How else could one name it, and, at the same time,

give any idea as to its nature? It is forming slightly distorted thymic tissue at the site of the thymus, and its appearance is immediately recognizable as thymic. It is the largest tumor of this organ that we have been able to find reported, its nearest rival being that of Crosby,[7] which weighed 1,975 gm. and measured 30 × 13 × 12 cm. Most of these tumors weigh in the neighborhood of 50 gm. No case that we can find has survived operative removal of a thymoma more than a few days, which again makes our case unique. Most thymomas, usually

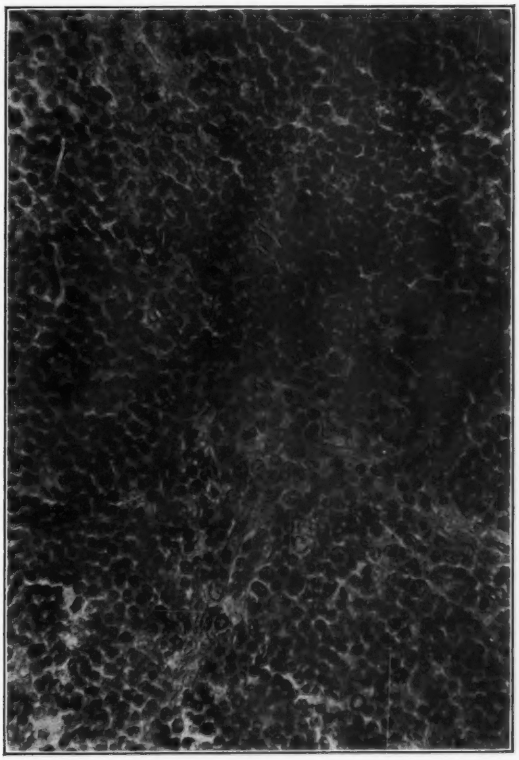

Fig. 7.—Silver impregnation of a section to show the interlobular reticulum network. (Foot and Foot. ×850.)

malignant, so involve the neighboring structures as to make excision impossible, or extremely difficult. Our case showed sharp encapsulation. It seems incredible that if these well-differentiated tumors have any functional activity (as indicated by their frequent association with myasthenia gravis), our patient showed nothing referable to thymic dysfunction, unless his easy fatigability might be considered as due to this cause, rather than to the mechanical interference with heart action and respiration.

GENERAL CONSIDERATION OF THYMIC TUMORS

Since 1926, when one of us (Foot[8]) last reviewed the literature on thymic tumors, an increasing number of these has been reported up to 1932, when Crosby[7] was able to find the respectable total of 166, 144 of which he tabulated in his excellent paper under two group headings: sarcomatoid and carcinomatoid. In his tables he gives the names of the reporters, age, sex and clinical data on the cases, with reference data, thus bringing the subject up to date in a most complete fashion

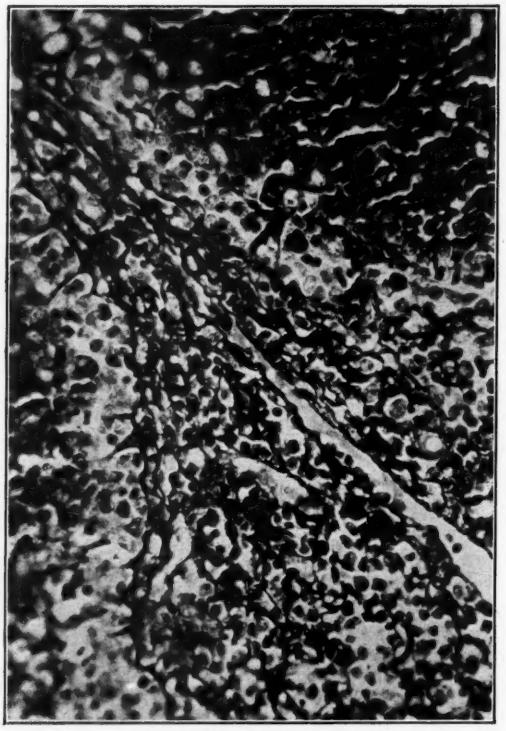

Fig. 8.—This demonstrates the most undifferentiated area found. Note the large number of mitotic figures. Some of these dark spots may represent pyknotic degeneration of nuclei. (Giemsa. ×800.)

and contributing a very interesting case of his own. Symmers[9] in the same year reviewed his own formidable series of 25 collected cases at Bellevue Hospital, New York City. Norris[6] has collected and tabulated data on 35 cases of myasthenia gravis, connected with thymic tumors in about 50 per cent of the cases, the other half showing simple hyperplasia, or no morphologic changes of note. He reports a tumor very similar to ours. His paper is a valuable contribution on the nonmalignant group of thymic tumors.

The more cases one finds reported, the more types of "thymoma" are encountered. First called by this name by Grandhomme[10] in 1900, the tumor has been the subject of much discussion; the term has come in for much criticism and the fact that we are still disputing over the origin of the small thymocytes leaves us undecided as to whether the lymphoid types of the tumor of the thymus are really lymphocytic or not.

As to this term "thymoma," it would seem that we can do no better in the case of those forms of the tumor that contain every, or nearly every, element of the gland. They may be so disarranged as to preclude the idea that this is simple hypertrophy or hyperplasia, or they may be somewhat disguised. The fact remains that they are there and that they definitely differ from one another and show no uniformity, in which case one is forced to fall back upon the name of the organ in naming the tumor. Again, in dealing with growths composed of lymphoid cells of doubtful origin, we are forced to resort to a looser term until this point of histogenesis has been settled.

The fact that several tumors of this latter category have been reported in connection with a concomitant lymphoid leucemia (Friedlander and Foot,[11] Symmers[9]), leads one to believe that these cells are of mesodermal, rather than entodermal origin. In the absence of other lymphoid hyperplasia in the lymph nodes or spleen, it seems hard to conceive that these cells come from anywhere else than the tumor, which shows a marked increase in the number and proliferative activity of small lymphocytoid elements. It seems equally hard to swallow the hypothesis that entodermal cells could circulate and mutiply in the blood stream in such numbers as to constitute a "lymphoid leucemia" indistinguishable from the ordinary type. Lymphocytes found in the blood under such circumstances need no explanation, as they are in one of their usual habitats; to claim that they are entodermal thymocytes requires rather more elaborate hypotheses for its elucidation than the average man will accept. In spite of this, Margolis[12] has criticized this viewpoint in an excellent paper, although he brings forward very little in support of his criticism. In all fairness, it must be admitted that some tumors show slight differences in the appearance of their small cells from that of lymphocytes that may cause the proponents of the lymphocytic theory transient uneasiness; this is true in such cases as that of Goyle[3] and others.

On reviewing the literature, one is struck with the fact that there are apparently more types of thymoma than was earlier supposed; as cases have been added to the list, new forms have appeared. This is particularly evident in the "perithelial" group. It would be well to sum up the results of our recent review.

Nonmalignant Thymoma.—Aside from thymic rests, either in situ or ectopic, and of the choristoma variety, there are reports of tumors

composed of disarranged though otherwise practically normal thymic tissue. The thymic reticulum cells tend to lie peripherally, the thymocytes centrally in the lobules, reversing the normal situs. Thymic corpuscles are usually present and have the characteristic appearance. The eosinophile thymic cells are apparently somewhat in eclipse in these tumors, although a careful search may reveal their presence.

Malignant Thymoma.—1. Thymocytic or lymphocytoid type: These are composed of small, round cells with deeply-staining nuclei; Ewing[13] believes that they are darker than lymphocytes. Hassall's corpuscles may or may not be present.

2. Large-celled or lymphoblastic type: These are made up of large, rounded to ovoid cells resembling lymphoblasts or primitive thymic reticulum cells. Thymic corpuscles may or may not be found.

3. Thymic reticulum cell type: The cell of this group closely resembles the entodermal thymic reticulum cell, and the group is probably the largest and most frequently occurring of the malignant thymomas. It has been reported in man as well as in several animals, notably the chicken, cow, horse and sheep. Feldman[14] states that the cow seems to be most frequently affected. The cells of these tumors are more pleiomorphic, ranging from spheroidal to spindle-shaped, or multipolar and stellate. They may often form ductlike structures that are so definitely epithelial in their appearance that Schmincke[15] included these tumors in his new group of "lymphoepitheliomas." Thymic corpuscles are variably present.

4. Perithelial type: This has been reported by Symmers[9] in eight of his 25 cases, and by Matras and Priesel[16] (Case 5) and Laas,[17] although the foreign authors do not refer to it as such. It shows definitely perithelial arrangement of radially directed fusiform cells about its vessels (be it noted, without interpolated areas of necrosis) and is quite characteristic in appearance. Thymic corpuscles do not play an important rôle in this tumor.

5. Granulomatous type: Symmers has collected five cases of Hodgkin's granuloma in the thymus, there may be others in the literature reported in connection with Hodgkin's disease and, therefore, missed in our review. The picture is that of Hodgkin's granuloma located in the thymus or at its site.

6. Epithelial, or carcinomatous type: These are tumors of elderly subjects and resemble epidermoid carcinoma as they are composed of innumerable thymic corpuscles in all stages of development and degeneration, thus creating a "pearly" appearance. The thymic reticulum cells may form radiating cords from such "pearls," which sometimes have ductlike arrangement, and, with a liberal admixture of lymphocytoid elements, may shift the picture toward that of the "lymphoepithelioma" of Schmincke.

7. Teratoid type: Here all the elements of the normal thymus are found to be undergoing abnormal proliferation, and they are often thoroughly disguised. The proliferation may be distributed through all the elements, or it may preponderate in one or two. In addition to quasi-normal components, duct- or glandlike structures may be found, or even solid medullary plugs of pale, rather delicate epithelium. These tumors seem to be more frequent in young individuals. Margolis[12] suggests that ducts of this sort might be found in normal thymus and indicate the presence of rests that might later give rise to tumors of this sort. His reported tumor was quite rich in such structures, which were less prominent, though present in a case reported by Foot and Harrington.[18]

SUMMARY

An essentially nonmalignant tumor of the thymus, weighing 2,235 gm. and successfully removed from a boy of thirteen, is reported from the clinical and pathologic standpoints. Its pathologic features are discussed, and the recent literature on the subject reviewed and commented upon.

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BRONCHORRHEA

Its Occurrence in a Case of Bronchiogenic Carcinoma

Theodore L. Bliss, M.D.
Akron, Ohio

THE term bronchorrhea apparently has disappeared from use in this country. The only reference revealed by a rather thorough search of the literature was in Williams'[8] *System of the Practice of Medicine,* published in 1841. In his book, Williams defined bronchorrhea as "an excessive expectoration of a pint or more of clear, slightly viscid, frothy secretion, which appeared to be the natural mucus diluted with a considerable addition of the watery part of the blood, with some portion of its saline matter." He noted the occurrence of bronchorrhea in cases of asthma, hay fever, cardiac decompensation, occasionally miliary tuberculosis, and in one case—"in connexion with malignant disease of the bronchial glands, which seemed to press on several of the great pulmonary blood vessels." Williams considered bronchorrhea to be a disorder in the function of secretion of the bronchi, which was independent of an inflammatory process, and he did not apply the term to the excessive purulent sputum which may occur in cases of bronchiectasis, lung abscess, pulmonary tuberculosis, and other inflammatory diseases of the bronchi and lung.

So far as I am aware, the term bronchorrhea is not used in America today to describe the copious production of purulent sputum. In the European literature, however, bronchorrhea is commonly used to describe excessive sputum which is purulent and is caused by inflammatory diseases of the bronchi and lungs, particularly bronchitis, bronchiectasis, and lung abscess. Casotto,[1] Jacquet,[4] Moulonguet,[5] and others have described such cases in articles published under the title of bronchorrhea.

In the present study I employ the term bronchorrhea, as Williams did, to describe the abundant outpouring of an apparent bronchial transudate, and not to indicate an excessive bronchial exudate resulting from inflammatory disease, as European authors use the term. I have been unable to find in the literature the report of a case of bronchiogenic carcinoma in which bronchorrhea has been an outstanding symptom.

CASE REPORT

The patient was referred to me by Dr. W. T. Fenker of Sandusky, Ohio, for a chest examination, and I am indebted to him for the use of his data on the case, which include the autopsy findings. The patient was a white man, aged forty-seven years, who was a chemist in a fertilizer plant, which exposed him to chemical irritants during his working hours. The prevailing chemical fumes were sulphur dioxide

and stripped ammonia, with exposure to heavy and short-term doses of the fumes of nitrous oxide and sulphur chloride. His mother died of cancer at the age of sixty-five years. Otherwise, the family history was not significant. The patient had had no serious illnesses in the past. He had noted dyspnea on moderate exertion for some time, but this had not increased during the last year. The patient's history and the family history were negative for allergic manifestations.

He began to be troubled by an unproductive cough in July, 1934. In November, 1934, he had an acute right-sided pleurisy without effusion, following which the

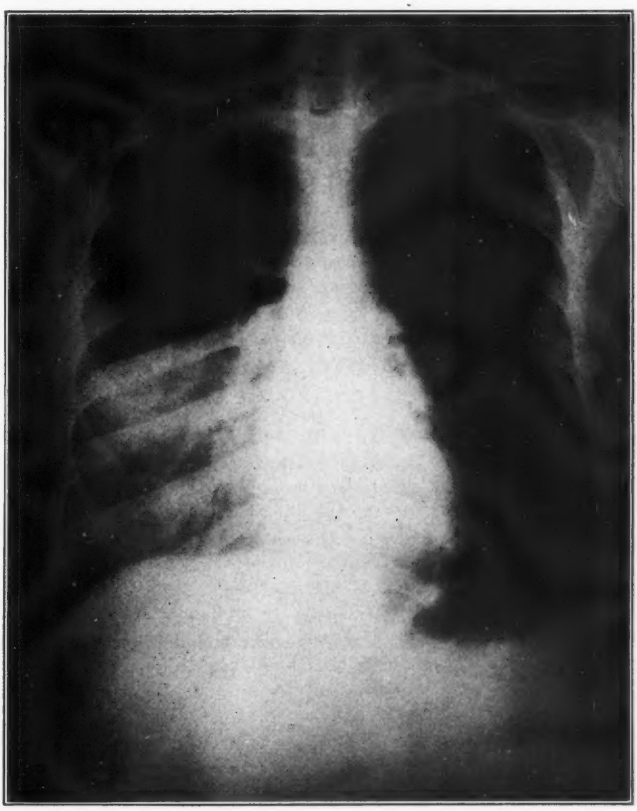

Fig. 1.—Chest roentgenogram. Increased density in the area corresponding to the right middle and lower lobes. In the lower lobe is a less radiolucent area.

coughing was aggravated and he began to raise small quantities of mucoid sputum. In December, he had an acute illness which lasted for four days, accompanied by fever as high as 102° F. and general malaise. Following this incident, his strength and energy were decreased, his cough became more severe, and he began to raise increasing quantities of a thin serous secretion. There was no history of hemoptysis or blood-streaked sputum.

Physical examination in January, 1935, showed that the patient was emaciated, but normally developed. His weight had decreased from 138 to 131 pounds in the previous six weeks. The systolic and diastolic blood pressures were 100 and 76; the

pulse rate was 88, and the temperature 98.4° F. There were no palpable glands in the cervical, supraclavicular, axillary or inguinal regions, and the fingers were not clubbed. The thorax was symmetrical and was flattened anteriorly. Expansion of the right lower hemithorax was restricted, and over this area a friction rub was palpable, tactile fremitus was decreased, and the percussion note was dull to flat. On auscultation there was a loud friction rub which extended up to the angle of the scapula, and the bronchovesicular breath sounds were distant. Coarse posttussic râles were heard in the right interscapular area. The apex of the right lung and the entire left lung gave normal physical signs.

In the six-foot chest roentgenograms (Fig. 1), taken in the posteroanterior and right lateral positions, there was some increased density in the region corresponding to the right, middle and lower lobes, and the sulci between the upper and middle, and the middle and lower lobes, could be delineated. There was no characteristic evidence of pleural effusion. In the region corresponding to the right lower lobe, there was an area of irregular outline that was comparatively less radiolucent which measured approximately 2 cm. in diameter. The overlying and surrounding opacity somewhat obscured the margins of this circumscribed area and it could not be outlined distinctly. The right upper lung field and the left lung appeared normal, and there was no evident widening of the mediastinal shadow.

Urinalysis yielded normal findings. The blood Wassermann and Kahn tests were negative. The hemoglobin was 106 per cent by the Newcomer method, the erythrocytes numbered 5,120,000, and the leucocytes 8,800.

The bronchial secretion was watery and limpid; it never contained pus or blood, and a foul taste or odor never was noted. Bronchoscopy revealed that its source was the right lower lobe, which was the original site of the carcinoma. A slight cough was sufficient to expel the secretion, and when the patient was in the prone position, it literally flowed out. Although the quantity of bronchial discharge in a twenty-four hour period was not measured, careful and conservative estimates by the patient and Dr. Fenker placed the amount between 1,200 and 1,800 c.c.

Culture of the secretion on Sabouraud's medium failed to give a fungous growth. Repeated examinations of smears stained to demonstrate the *Mycobacterium tuberculosis* were negative. Unfortunately, chemical analyses of the secretion were not made.

By bronchoscopic examination, the lumen of the second division bronchi in the middle lobe and in both lower lobes could be visualized and showed no evidence of congestion, obstruction, distortion, dilatation, rigidity, mucosal thickening, ulceration, or newgrowth. The secretion was seen to well up solely from the right lower lobe, and it was so profuse that the examination was difficult technically.

The patient continued to lose weight and strength, and the bronchorrhea became progressively more profuse, but its character did not change. Subsequent roentgenograms of the chest revealed an increase in the size of the less radiolucent area in the right lower lobe, and the appearance of a linear infiltration radiating from the hilum into the right upper lobe. Shortly before death, a similar infiltration into the left lung was observed in the roentgenogram.

In July, 1935, a second bronchoscopic examination was made elsewhere. The report follows: "The carina is sharp and the mucous membrane shows no congestion. No purulent secretion is present but both bronchi, particularly the right one, contain large quantities of watery secretion which resembles esophageal material. The spurs on the left are normal. On the right there is marked narrowing of the lower lobe bronchus, immediately below the level of the middle lobe spur. This stenosis is caused by a smooth pushing in of the medial bronchial wall and shows no congestion or ulceration." Fluoroscopy of the esophagus with an opaque mixture revealed no evidence of a fistula between the esophagus and the trachea. Bronchog-

raphy with iodized oil showed no evidence of bronchiectasis and no gross obstruction to any of the bronchial divisions, although there was some puddling of the opaque medium a short distance below the origin of the right lower lobe bronchus.

The patient died in August, 1935, thirteen months after the onset of the symptoms. Dr. Fenker performed the autopsy and reported that there were no significant findings other than in the lungs. He found no metastasis, but did not open the cranial vault, since permission to do this had been refused. The patient, however, had no symptoms indicative of a cerebral metastasis prior to his death. The right pleural cavity was obliterated completely by dense, firm adhesions. The left pleural cavity contained a few easily separated adhesions. Both leaves of the diaphragm were free from invasion. The right lung was replaced by a grayish white mass, which was extremely friable, except for a small atelectatic area at the apex. The mass did not invade the parietal pleura, contained no areas of necrosis or liquefaction, and did not exude pus when sectioned. The same type of growth replaced approximately one-third of the left lung adjacent to the hilum, but did not extend to the periphery of the lung. The pericardium, the heart, and mediastinal structures were not invaded by the mass, and Dr. Fenker did not find any mediastinal lymph nodes which appeared to be involved upon gross examination.

Microscopic sections of the growth were diagnosed as a papillary form of bronchiogenic adenocarcinoma and showed no evidence of mucin production. Permission to remove other tissues for microscopic sections had been refused.

COMMENT

The mechanism which produced the bronchorrhea in this case merits discussion.

Edwards,[2] Rabin and Neuhof,[7] and others have described the sputum which is expectorated when bronchiogenic carcinoma obstructs a large bronchus producing bronchiectasis, or when the tumor tissue becomes necrotic and secondarily infected. Under these circumstances the patient may expectorate large quantities of sputum, but it is purulent and often has a foul odor. In the case I am reporting the sputum was never purulent and a foul odor never was noted. In addition, the autopsy did not reveal bronchiectasis, or necrosis of the tumor.

The expectoration of a nonpurulent sputum by patients with bronchiogenic carcinoma has been reported by Edwards. A number of his cases had a definite amount of regular expectoration which was generally of a mucoid frothy type. The amount of the sputum apparently was small, and did not constitute an outstanding sign. Edwards did not discuss the mechanism of its production.

The nonpurulent character of the sputum expectorated by patients with pulmonary edema is well known. Pulmonary edema may occur in patients who have nephritis, coronary sclerosis, cardiac decompensation, and sometimes, in patients following thoracocentesis. Mumme[6] reported three cases of "sero-albuminose" expectoration which followed the too rapid aspiration of a pleural effusion. He used the term "sero-albuminose" expectoration rather than the term bronchorrhea to describe the sputum. In one of his cases, the patient expectorated 750 c.c. of sputum in twenty-four hours. Mumme believed the expectoration was

a transudate due to pulmonary edema, and was not a portion of the pleural effusion which had reached the trachea through a pleurobronchial fistula or through a transgression of the pleura. The sero-albuminous expectoration was frothy, was usually blood-tinged, and was rich enough in fibrin to become clotted. In the cases Mumme reported, an irritating cough preceded the onset of the edema, and during the attack the patients became dyspneic and cyanotic, and had a weak and thready pulse. The entire lung on the aspirated side was involved, and moist bubbling râles were heard over that side of the chest. Mumme discussed a number of factors which play a part in producing pulmonary edema when a pleural effusion is aspirated too rapidly. He pointed out that the decrease in the intraalveolar air pressure during inspiration sucks blood into the capillary bed, which is increased in size during inspiration. If, in addition, aspiration has rapidly decompressed the lung, hyperemia is the result. During the ensuing expiration the left auricle is embarrassed by the increased volume of blood forced upon it, and back pressure in the capillary bed will occur. Another factor which tends to produce edema according to Mumme is the absence of an epithelial lining of the alveolar wall. He pointed out that the only effective barrier to the escape of blood serum is the endothelial wall of the capillary. Still another factor that Mumme discussed was the rôle of the complex and sensitive vasomotor nerve supply of the lung capillaries. When they are irritated, active vasodilatation of the capillary bed results. He believed that rapid decompression of the lung could act as an irritation.

In the case I am reporting, pulmonary edema did not appear to be the cause of the bronchorrhea. The sputum was not characteristically frothy, or blood-tinged; there was no evidence of generalized moisture in the lung on physical examination, and there were no clinical signs of cardiac embarrassment, all of which are present in cases of pulmonary edema. In addition, the first bronchoscopic examination showed that the secretion came fr m a localized area of the lung. Mumme, however, discussed two factors that help to produce pulmonary edema, which may have helped to produce the bronchorrhea in the case I am reporting. They are irritation of the vasomotor nerves by the tumor, and the absence of an epithelial lining in the lung alveolus.

The bronchorrhea could have been a product of the tumor. However, the microscopic sections of tissue obtained at autopsy indicated that the carcinoma was not producing mucin. This would justify the conclusion that the bronchorrhea was not a product of the tumor in this case.

Hyperactive secretion of the bronchial mucosa due to irritation of the tumor offers a possible explanation for the bronchorrhea. It is doubtful, however, that mechanical irritation, acting on a limited area of bronchial mucosa, could produce such large quantities of secretion. The possibility of hyperactive secretion of the bronchial mucosa due to allergic hypersensitiveness must be considered, even though allergic

manifestations were not elicited in the history of patient or his family, and a possible causative allergen was obscure. Fisher[3] has suggested the theory of autosensitivity to an allergen which was a product of the tumor metabolism.

The only other suggestion which I can offer to explain the bronchorrhea in this patient is on the basis of lymphatic obstruction. If the carcinoma had obstructed the deep or peribronchial lymphatic channels, the overflow of lymph could have escaped through the bronchial lumina since it is known they were patent. If, in addition, the superficial or subpleural lymphatic channels had been obstructed by the fibrinous pleurisy which the patient had had over the right, middle, and lower lobes, lymph could not have drained in that direction. The increase in the quantity of fluid expectorated which paralleled the extension of the carcinoma, gives some support to this hypothesis.

I do not know whether any diagnostic significance could be attributed to the bronchorrhea in this particular case. It would seem reasonable, however, to consider bronchorrhea of unilobar origin as suggestive of bronchiogenic carcinoma, when it is not attributable to another cause.

SUMMARY

1. A case of bronchiogenic carcinoma with the autopsy findings is reported.

2. The unusual feature of the case was the excessive quantity of clear serous secretion which the patient expectorated. One possible explanation of this excessive secretion is that the carcinoma, which originated in the periphery of the lung, obstructed the deep peribronchial lymphatic channels, and that, since the major bronchi were not occluded, the overflow of lymph escaped in this manner.

3. This bronchial secretion has been called bronchorrhea, according to a definition by Williams in 1841, who stated that excessive discharge of this type connotes a noninflammatory lesion of the lung.

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THE RIGHT MIDDLE LOBE AND THE LATERAL ROENTGENOGRAM*

DONALD S. KING, M.D.
BOSTON, MASS.

IN THESE days of highly successful thoracic surgery, the correct diagnosis of pulmonary lesions and their accurate localization is of increasing importance. In such an investigation the x-ray findings are of the utmost value, and of the newer developments in x-ray study, the "lateral film" has proved to be one of the most useful. The lateral view of the chest is of especial importance to the surgeon in determining the presence or absence of interlobar effusions and in the localization and diagnosis of disease in the right middle lobe. It is not possible within the limitations of this paper to take up the whole problem of the interpretation of the lateral x-ray film, to discuss adequately the problem of interlobar effusion, or to illustrate all the disease processes which may be confined to the middle lobe. Many of these points are elaborated in another article published with Dr. Aubrey O. Hampton[1] of this hospital. The present article will emphasize only the most important practical points which have been of help at the Massachusetts General Hospital in the past few years. Frequent reference will be made to the x-ray films reproduced in the former publication, but, except in an occasional instance, they will not be republished.

INDEFINITE SHADOWS IN THE POSTEROANTERIOR ROENTGENOGRAM

Roentgenologists often describe areas of indefinite density in the lower third of the right lung field as due to "perihilar thickening, peribronchial fibrosis, pleuropericardial disease, increased lung markings, thickened interlobar pleura," etc. Definite diagnosis is impossible in these cases with the ordinary posteroanterior film, but with the lateral film, the process can often be localized in the middle lobe. Such a case is illustrated in Fig. 1. Here there was atelectasis of the right middle lobe caused by the obstruction of its bronchus by a blood clot. At bronchoscopy the blood clot was removed, and roentgenograms then showed a complete reexpansion of the middle lobe.

KREUZHOHLSTELLUNG (EXTREME LORDOSIS)

In the past, particularly in Germany, roentgenologists[2, 3] who have not been satisfied with the ordinary posteroanterior film have learned that

*From the Massachusetts General Hospital, Boston, Massachusetts.
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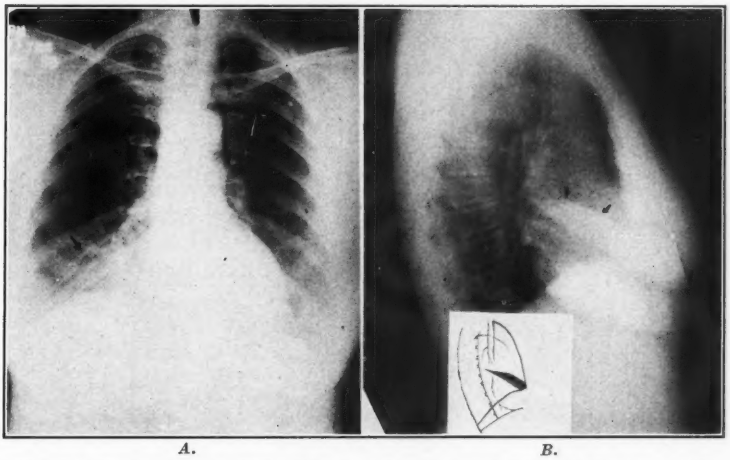
roentgenograms taken with the patient in the position of extreme lordosis have often been sufficient to localize the disease in the middle lobe. These films are superior to the stereoscopic films for this purpose, but are not in most cases as satisfactory as the lateral films. Fig. 2 gives the roentgenograms of a case of carcinoma of the middle lobe bronchus producing atelectasis of this lobe. Fig. 2*A*, the ordinary posteroanterior film, shows the sharp upper border of the area of density which at this level is usually sufficient to localize this disease in the middle lobe; Fig. 2*B*, taken in the position of extreme lordosis, brings out the localization of the process more clearly than 2*A*; but Fig. 2*C*, the lateral film, localizes the process definitely in the region of the middle lobe.

Sometimes, however, the film taken in extreme lordosis gives a cleaner-cut shadow than the lateral film. Fig. 3 demonstrates such a case. Fig.



A. *B.*

Fig. 1.—Blood clot in middle lobe bronchus. *A*, Indefinite shadow in posteroanterior film. *B*, Lateral film bringing out middle lobe atelectasis. (Courtesy Am. J. Roentgenol.)

3*A*, the ordinary posteroanterior film, shows a lesion close to the right border of the heart; in Fig. 3*B*, the lateral film, there is only indefinite haziness in the region of the middle lobe; but in the film reproduced in Fig. 3*C*, taken in the position of extreme lordosis, the middle lobe lesion is clearly demonstrated. Fig. 3*D*, with lipiodol in the bronchial tree, shows the obstruction of the right middle lobe bronchus which is responsible for the partial collapse of the middle lobe.

TRIANGULAR AND RECTANGULAR SHADOWS IN THE MIDDLE LOBE

In the past, it was believed that a diseased middle lobe always cast a triangular shadow in the lateral roentgenogram, but closer observation shows that the shape of the area of density in the x-ray film depends

on what portion of the middle lobe is involved. A brief anatomic study of the middle lobe will clarify this point. Fig. 4*A* shows the

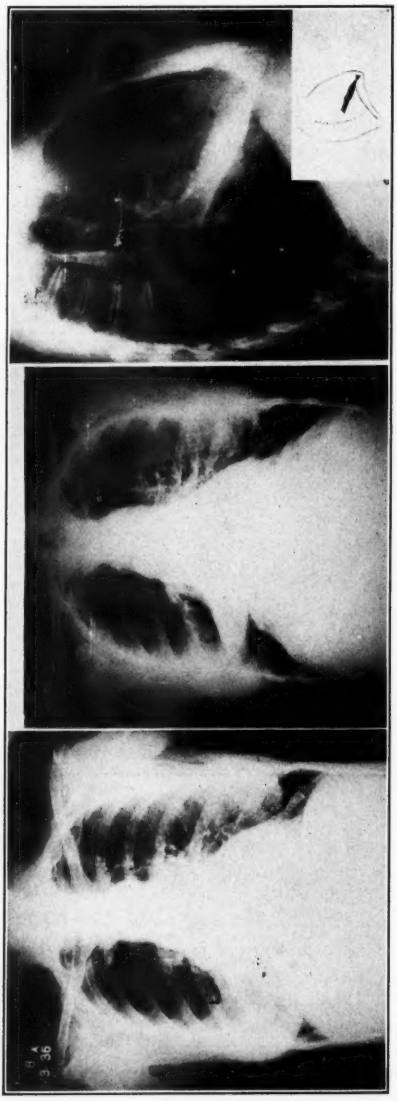


Fig. 2.—Carcinoma of the middle lobe bronchus with atelectasis of the middle lobe. *A*, Posteroanterior view showing sharp upper border of the middle lobe. *B*, Middle lobe atelectasis as shown in extreme lordosis (Kreuzhohlstellung). *C*, Middle lobe atelectasis as shown in the lateral view.

lateral aspect of an inflated normal right lung. The fissures are, of course, exaggerated, but this picture brings out clearly the essentially triangular shape of the lateral margin of the right middle lobe. Fig. 4*B* demonstrates the medial aspect of this lobe, and it is more nearly

rectangular in shape. It is reasonable then to assume that if only the lateral portion of the lobe is involved, the x-ray shadow will be triangular; if the medial portion alone is involved, the shadow will be rectangular.

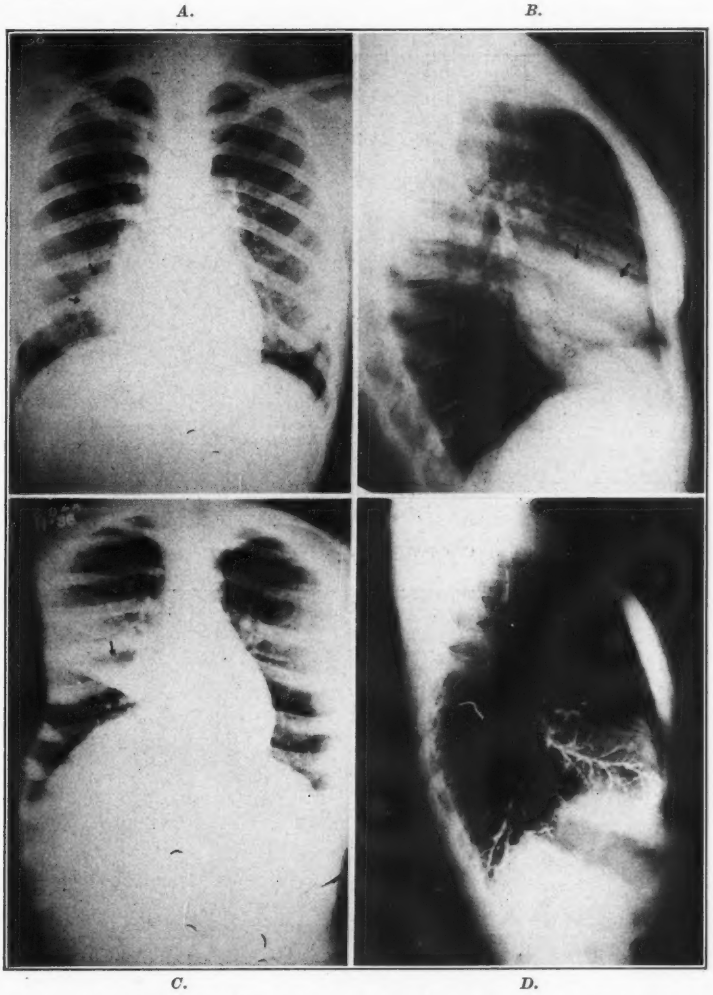
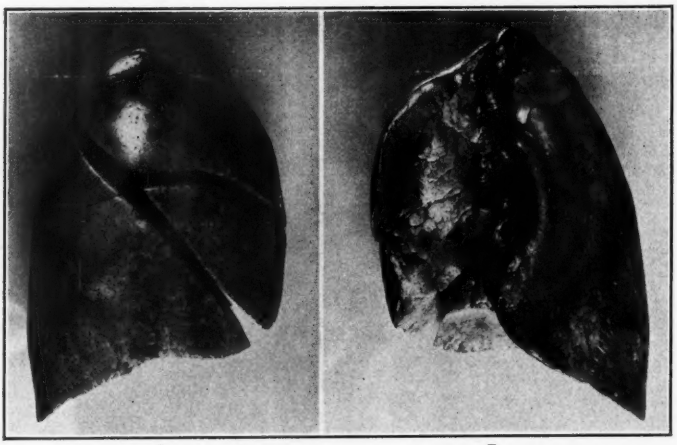


Fig. 3.—Obstruction of middle lobe bronchus with atelectasis. *A*, Posteroanterior view. *B*, Lateral view showing indefinite density in the region of the middle lobe. *C*, Middle lobe atelectasis shown in extreme lordosis (Kreuzhohlstellung). *D*, Ostruction of middle lobe bronchus shown by lipiodol.

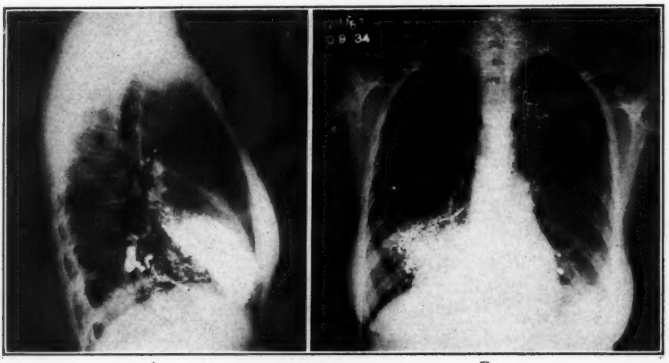
Fig. 5 illustrates the essentially rectangular shape of the medial surface of the middle lobe, as brought out by a lipiodol filling concentrated mostly in the medial portion of the lobe. This shadow is different

from the triangular shadow which is supposed to be typical of any involvement of the middle lobe. A rectangular x-ray shadow, due to a pathologic process in the middle lobe, was reproduced in the previous article. In this case, there was carcinoma in the right middle lobe bronchus, and at autopsy, marked edema was found in the medial por-



A. *B.*

Fig. 4.—Inflated normal lung. *A*, Lateral view showing triangular shape of lateral aspect of the middle lobe. *B*, Medial view showing rectangular shape of the medial aspect of the middle lobe. (Courtesy Am. J. Roentgenol.)

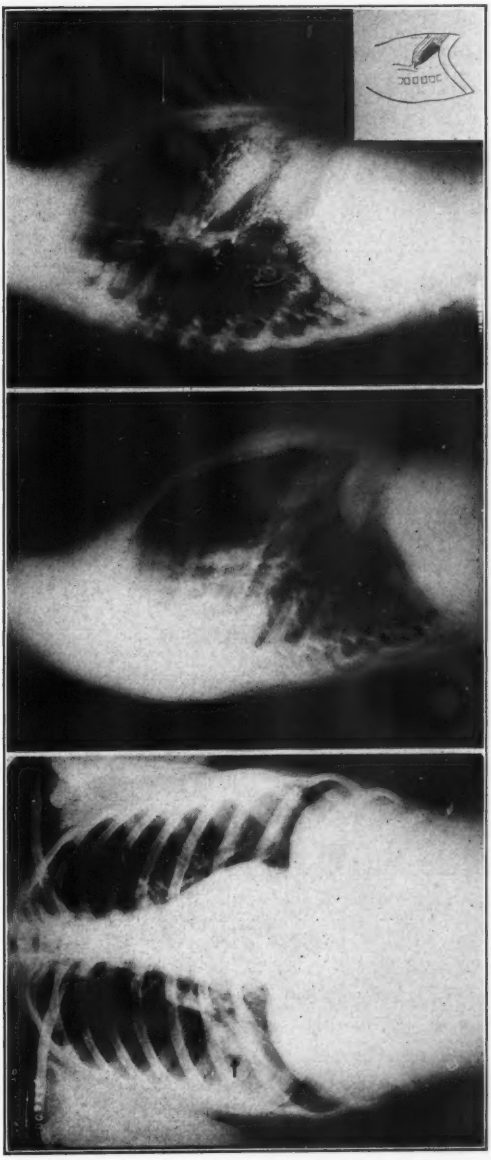


A. *B.*

Fig. 5.—Normal middle lobe—lipiodol injection. *A*, Lateral view showing rectangular shape of the middle lobe—medial portion. *B*, Posteroanterior view—most of lipiodol in medial portion of right middle lobe. (The bronchiectasis is in the left lower lobe.)

tion of the middle lobe which was responsible for the rectangular shadow produced in the roentgenogram.

In the beginning, we inclined toward the idea that the triangular or rectangular shape of x-ray density was dependent upon the distribution



A. *B.* *C.*

Fig. 6.—Obstruction of one of two main branches of the middle lobe bronchus. *A*, Posteroanterior view. *B*, Lateral film showing partial atelectasis of the middle lobe. *C*, Lipiodol filling showing obstruction.

of the two main branches of the bronchus to the right middle lobe. We were assuming that one of the branches supplied the lateral half of the lobe, while the other supplied the medial half. On this assumption we thought it probable that pathologic changes involving the lateral branch would produce a triangular shadow, and that changes involving the medial branch would produce a rectangular shadow. A study of the anatomy of the bronchi, however, does not allow the acceptance of this explanation. Students of the anatomy of the middle lobe bronchi agree in general that the two branches supply essentially the anterior

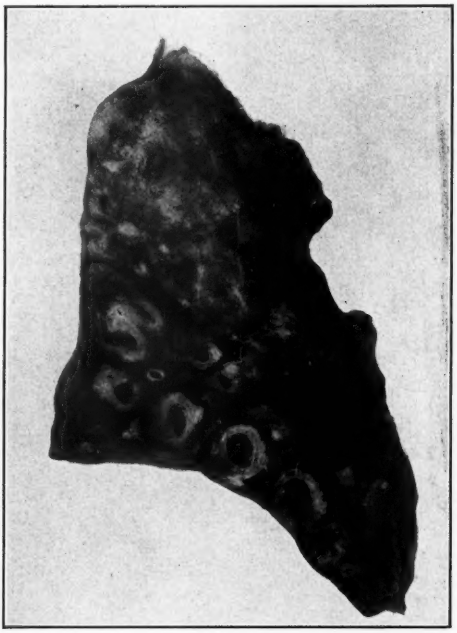


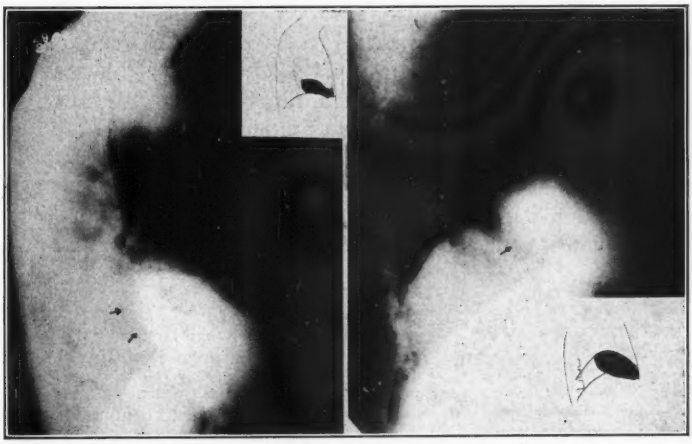
Fig. 7.—Surgical specimen showing bronchiectasis in one-half of the right middle lobe. (Courtesy Dr. Edward D. Churchill.)

and posterior portions of the lobe, rather than the lateral and medial. One branch goes superiorly and anteriorly, while the other is primarily posterior and lateral.[4, 5] An article by Davis[6] brings out a point which should be kept in mind, namely, that there is probably marked difference in the distribution of the main branches of the right middle lobe bronchus. Glass and Kramer,[7] however, report 80 cases studied with only 15 per cent anatomic variation.

An interesting case in our series showed obstruction of unknown etiology in one branch of the middle lobe bronchus. The x-rays of this case are shown in Fig. 6. The posteroanterior view illustrates a lesion

which one would assume to be in the middle lobe, and the lateral roentgenogram localizes the area of density in the antero-inferior portion of the right middle lobe. The lipiodol filling (Fig. 6*C*) shows that the obstruction involved only the inferior branch of the middle lobe bronchus, and the triangular area of atelectasis in the portion of the middle lobe supplied by this branch is superimposed upon a rectangular area of lipiodol infiltration, such as was shown in Fig. 5.

Fig. 7 shows the surgical specimen of a case of bronchiectasis which involved only one-half of the right middle lobe, the other half being normal. The limitation of the diseased process to half of the lobe was due to the fact that only one branch of the middle lobe bronchus, with its subdivisions, was involved in the bronchiectatic process.



A. *B.*

Fig. 8.—Overlap shadow of the heart and high right diaphragm. *A*, Shadow of the right diaphragm not seen. Spindle-shaped shadow due to overlapping heart and high right diaphragm. Wrongly interpreted as interlobar effusion. *B*, Both diaphragms now plainly seen and overlap shadow obvious. (Courtesy Am. J. Roentgenol.)

"SPINDLE-SHAPED SHADOWS" OF INTERLOBAR EMPYEMA

It was a search for cases of true interlobar empyema that first stimulated our interest in the lateral roentgenogram. Surgeons and roentgenologists speak so freely of the occurrence of this condition and of a typical roentgenogram which is supposed to go with it, that we were much surprised to discover not a single case in the records of the Massachusetts General Hospital of pure interlobar empyema which had been diagnosed by the roentgenologists and proved by operation or autopsy. Definitely proved interlobar empyema was either part of a generalized empyema or was secondary to pulmonary abscess. We still believe that cases of primary interlobar empyema without pus in the pleural cavity or abscess in the lung occur, but the fact remains that we

have not, after careful search, been able to find any in this hospital. In one of our cases, there is shown an essentially rectangular shadow in the region of the middle lobe which was caused by middle lobe abscess and interlobar empyema. On this rectangular area is superimposed an indistinct triangular shadow which may be that of the lateral border of the middle lobe.

"OVERLAP SHADOW"

Because of the importance of this point for the thoracic surgeon, we are again reproducing Fig. 8, the roentgenogram from a postappendectomy case. Following operation there was intermittent elevation of temperature and foul sputum. In the lateral roentgenogram, the "spindle-shaped shadow" was originally interpreted as proving interlobar empyema. It will be noted in this film that the posterior half of the right diaphragm is not visible, and at the present time, such an observation would undoubtedly put us on the right track; but at the time this film was taken we did not realize the importance of visualizing both leaves of the diaphragm in the lateral film and were led astray by the finding of what we thought to be an unusual shadow.[8] Fig. 8*B* shows the same "spindle-shaped shadow" in a film of the same patient taken later in the course of the disease. Here it is obvious that this shadow in the anterior half of the chest is due to the overlapping of the high right diaphragm and the posterior border of the heart.

TUMORS OF THE LIVER

In these days when so many lung tumors have been shown to be operable, it is important that the surgeon be sure that he is not confusing a tumor below the diaphragm with one above it. The lateral roentgenogram has to be carefully studied to rule out such a possibility.

VARYING DEGREES OF MIDDLE LOBE ATELECTASIS

All degrees of atelectasis may be present in the middle lobe where there is bronchial obstruction from different causes, or where bronchiectasis is present. In many instances, the middle lobe is so completely collapsed that it is spoken of as a "pancaked" middle lobe. We have found that most shadows in the lateral roentgenogram that were formerly interpreted as due to the thickening of the interlobar septa are in reality due to complete or partial atelectasis of the middle lobe itself. In these cases, bronchoscopy or lipiodol injection demonstrates the obstruction of the main bronchus to the middle lobe or one of its branches. Such a case is illustrated in Fig. 6 above.

OTHER PATHOLOGIC CONDITIONS OF THE MIDDLE LOBE

Besides bronchiectasis, carcinoma, and abscess of the middle lobe as illustrated above, our observations have shown tuberculosis, lobar pneumonia, infarct, fibrosis, and emphysema limited to this lobe. These films

cannot be reproduced here. The lateral roentgenograms also frequently show displacements of the middle lobe and confusing shapes due to adhesions of the lobe to the anterior chest wall or diaphragm. A study of the septa bounding the middle lobe is often helpful.

Certain obvious values of the lateral roentgenogram have not been mentioned in this paper. The first use of a film taken in this position was to localize lesions anteriorly, posteriorly, or at the root of the lung. This point is of special diagnostic value in differentiating a neurofibroma, originating in the sympathetic chain and, therefore, usually located posteriorly from a dermoid cyst which is commonly located anteriorly. The lateral film also helps to determine the relation of a pathologic lesion to the ribs, sternum, and so forth. Occasionally, the lateral roentgenogram will show that a process which in the posteroanterior film looks like a cavity with fluid level is actually a collection of air and fluid in the region of the interlobar fissure.

SUMMARY

1. The lateral roentgenogram may bring out definitely diagnostic shadows which are indefinite in the ordinary posteroanterior or stereoscopic films.

2. The lateral roentgenogram is usually, but not always, superior to the film taken in extreme lordosis (Kreuzhohlstellung).

3. Anatomically, the lateral border of the middle lobe is triangular in shape and the medial border rectangular. These different shapes are often shown in the lateral roentgenograms of normal or diseased middle lobes.

4. Empyema limited to the interlobar fissure rarely occurs except in association with pulmonary abscess. The "spindle-shaped shadow," said to be diagnostic of such a process, is rarely traced to this cause.

5. The overlap of a high right diaphragm and the posterior border of the heart gives a shadow often mistaken for the "spindle-shaped shadow" of interlobar effusion.

6. Primary tumors of the liver situated below the diaphragm must be considered in the interpretation of the lateral roentgenogram.

7. Varying degrees of atelectasis of the middle lobe are well shown in the lateral film and have in the past been misinterpreted as due to interlobar thickening.

8. The following pathologic processes have been shown to be limited to the middle lobe: bronchiectasis, tuberculosis, carcinoma, abscess, lobar pneumonia, infarct, fibrosis, and emphysema.

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EXPERIMENTAL PNEUMONECTOMY

LESTER C. THOMAS,* M.D., ALBERT BEHREND,* M.D., AND
FRANK C. MANN, M.D.†
ROCHESTER, MINN.

THE problems connected with experimental pneumonectomy have attracted the interest of investigators since the latter part of the nineteenth century. It is rather evident from even a cursory perusal of the literature covering this subject that the last word as to method and a full appraisal of the physiologic changes that occur following pneumonectomy have yet to be written. Recent reports by various surgeons of several cases in which pneumonectomy was successfully performed for bronchiectasis or malignancy lend added zest and practicability to such a study.

The purpose of this paper is to describe a method which has been found adequate for successful removal of a complete lung of the dog. The percentage of recoveries was high, making possible a sufficient amount of material to study the effects of pneumonectomy.

REVIEW OF THE LITERATURE

The first report of a series of cases in which experimental pneumonectomy was performed is that of Gluck who published his results in 1881. He removed an entire lung of each of six dogs and fourteen rabbits. With the exception of two of the rabbits, all these animals died of pleural or pericardial infections within ten days of the operation. In the same year, Block's results on a series of rabbits and dogs appeared, all the animals dying within a few days of pneumonectomy. Shortly thereafter appeared the encouraging work of Biondi. He injected animals with tuberculous material in the hope of producing localized pulmonary tuberculosis and then removed the infected lung. Seven of his animals recovered, four died. Zakhorevitch in 1890 treated the bronchial stump with iodoform, and of twenty-four animals so treated, nineteen recovered. Willard in 1891 obtained a successful result in one case by ligating en masse the hilus of the lung of a dog and then suturing the stump to the thoracic wound. Nine of ten dogs operated on by Murphy died despite an aseptic technique.

Meyer then obtained the best results up to that time when he performed pneumonectomy on twenty-one dogs with only four deaths. He attributed his good results to the use of a positive pressure apparatus for induction of anesthesia, and to an original method of treating the

*Fellow in Surgery, The Mayo Foundation.
†Division of Experimental Medicine, The Mayo Foundation.

bronchial stump. This consisted of crushing, ligating and inverting the remaining portion of the bronchus after the manner commonly employed today following appendectomy. About the same time (1909) Sauerbruch and Robinson reported the recovery of four of their thirty-eight animals which had been subjected to pneumonectomy. Halsted in 1909, emphasizing the necessity for strict aseptic precautions, performed twenty-one thoracotomies, infection developing in only one case. In 1911 Quinby and Morse operated on a series of thirty-eight dogs using Meyer's method. They resected the right lung of seventeen dogs, with three deaths, and the left lung of twenty-one dogs, with five deaths. Giertz in 1914 introduced a plug of fascia into the bronchus, as suggested by Henschen, with two deaths in ten pneumonectomies. Heuer and Dunn in 1920 and Bettman in 1924 experimented with various methods of handling the bronchial stump following removal of the lung of dogs. Heuer and Dunn had ten deaths in twenty-three experimental pneumonectomies, and only two of Bettman's twenty-seven animals recovered. Schlueter and Weidlein in 1926 performed four pneumonectomies using the Meyer method, with one death. In 1928 Joannides operated on fifty-four animals, performing complete or partial pneumonectomy. In most of these cases the lung was not completely removed at the bronchus, the stump being sewed over the remaining portion of lung. Nine animals died during operation, nine died of distemper following the operation, and in another case death was attributed to infection and leakage of the bronchial stump.

The experimental results of Adams and Livingstone in 1932 are probably the most satisfactory to date. They produced stenosis of the bronchus by preliminary application of 35 per cent silver nitrate through a bronchoscope, causing atelectasis of the pulmonary lobe to be removed, which was usually done a week later. Whether the method is of practical value in thoracic operations on man is yet to be shown. In their study, however, lobectomy was performed on twenty dogs without a death and pneumonectomy on eight dogs, the death of one of the latter being attributed to a faulty anesthetic apparatus.

Longacre recently has stressed the importance of preserving the blood supply to the bronchial stump.

EXPERIMENTAL METHOD

Dogs were used exclusively in this investigation. Blood counts were made both before and after operation. Roentgenograms of the chest were made as soon after operation as possible and thereafter at varying periods. All the animals were examined bronchoscopically under ether anesthesia at various times after operation so that the course of healing of the bronchus could be followed.

Technique.—Ether anesthesia was used in all operations, the anesthetic being administered by the combined method of intratracheal insufflation

and positive pressure. Strict asepsis was maintained throughout. The field of operation was cleansed with benzene and with two applications of 2 per cent iodine in ether. The animal was then draped with sterile sheets and towels.

The incision, 4 to 6 inches (10 to 15 cm.) long, was made through skin and fascia between the fourth and the fifth ribs. Sterile towels were clipped to the skin margins. The pectoral and latissimus dorsi muscles were then divided and spread. The intercostal muscle was cut during

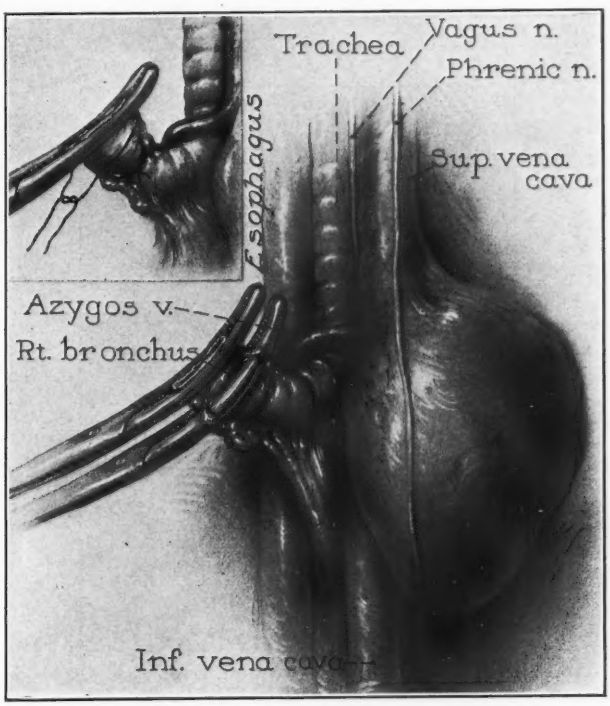

Fig. 1.—Showing the method of ligating the bronchial stump.

the expiratory phase of respiration so that the lung would not be injured. It was divided midway between the ribs, care being taken not to carry the incision too far anteriorly or posteriorly, so as to avoid injury to the internal mammary and intercostal arteries. A rib spreader was then inserted and opened as widely as possible, while warm moist towels protected the wound. This provided an excellent view of the pleural cavity and its contents. Positive pressure anesthesia is essential because, in the dog, the two pleural cavities are separated only by a thin veillike membrane which waves to and fro with respiration and is incapable of preventing the passage of either gases or fluids.

The pulmonary arteries and veins were then isolated, doubly ligated, and severed (Fig. 1). The bronchus was stripped of peribronchial tissue, care being taken not to injure the vagus nerve which is in close proximity to it. A piece of linen thread was then passed around the bronchus with the aid of an aneurysm needle as close to the tracheal bifurcation as possible, and not including the peribronchial blood vessels and lymphatics. It was tied firmly enough to occlude the lumen, but not so tightly as to crush the bronchus. A second ligature was placed about 1 cm. distal to the first, and this was tied more tightly and included all the peribronchial tissues (Fig. 2). Thus the first or prox-

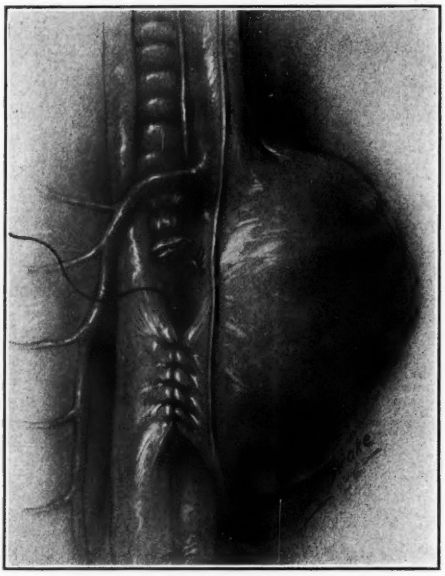

Fig. 2.—The pulmonary vessels and the right main bronchus have been individually tied, and burial of the stumps has begun.

imal ligature relieves the second or distal ligature from the strain of intrabronchial pressure and at the same time does not completely shut off the blood supply to the distal stump. In this way necrosis of the stump is prevented and healing most readily occurs (Fig. 3).

Clamps were then applied 1 to 2 cm. distal to the second ligature and the lung was extirpated. Fresh gauze was packed around the stump, the clamp was released, and closure was begun by means of fine silk thread on a small curved needle, the peribronchial tissues being utilized to oversew the stump. In all, three layers of suture line usually were used, and the bronchial stump was "pleuralized" much as the cervical

stump is peritonealized following hysterectomy (Fig. 4). The phrenic nerve, which is now in plain view as it crosses the pericardium, can be severed or not, as the operator desires.

Closure was obtained by pulling the separated ribs together by silk sutures thrown around them, according to the technique described by Beaver and Mann. The muscle layers were closed in the usual manner with a nonabsorbable suture. Before the last stitch was pulled tight, a hemostat was forced into the pleural cavity and spread while the anesthetist fully inflated the remaining lung, thus driving out residual air in the thoracic cage. The hemostat was then quickly removed at the height of inflation and the suture tied. This procedure lessened the

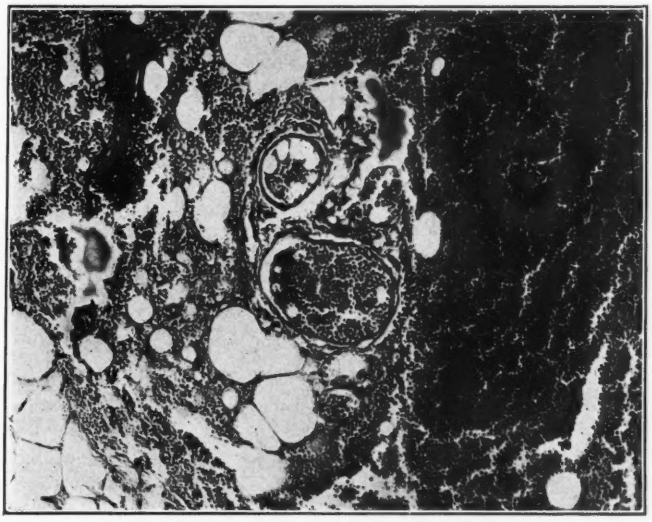

Fig. 3.—Some of the subtracheal blood vessels which are preserved to aid in the healing of the bronchial stump (×160).

negative intrapleural pressure and facilitated breathing. The skin was closed with linen thread, the incision covered with a strip of sterile gauze and the wound sealed with collodion. Very little postoperative care is required, the main consideration being to maintain the animal as quietly as possible.

Two other methods of bronchial closure were attempted, but both proved unsuccessful. In the first method a flat metal band, with rounded edges 1 cm. in width, was used to crush and close the bronchus. In the second a metal button of the same diameter as the bronchus was used to occlude the bronchial lumen after oversewing the button with flaps derived from the bronchial wall.

RESULTS

The results obtained with this simple technique have been very gratifying despite the fact that not a few of the dogs were lost after recovery from operation. These deaths are attributed to an epidemic of distemper and pneumonia which occurred in the kennels during the period of this investigation. The animals that survived were observed for periods ranging from a month to more than eight years.

Effect of Exercise.—The dogs which had been operated on were exercised daily with normal dogs as controls. At the finish of a long brisk run uphill and on the level they did not show any dyspnea or fatigue.

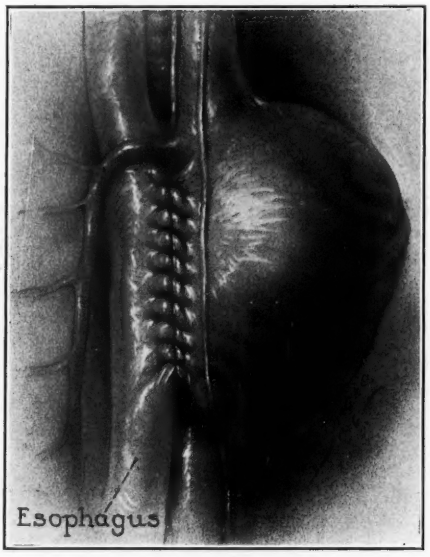

Fig. 4.—Showing appearance of mediastinum just before closure of thorax.

Roentgenographic Studies.—The changes that occur postoperatively in the thoracic cavity can be readily followed by means of roentgenographic examination. Films taken shortly after operation revealed little change from the normal. The heart was in the normal position, and the paralyzed left diaphragm showed no immediate change of position.

Films taken from three weeks to a month following operation showed that the heart was moving toward the wall of the chest on the side from which the lung had been removed and that the paralyzed diaphragm was rising into the pleural cavity. Films taken several months after operation revealed the empty space in the pleural cavity to be almost obliterated by the heart and remaining lung. The heart was adjacent to the thoracic wall and the diaphragm had risen high into the pleural

cavity. Whether the paralyzed diaphragm was pushed upward by intra-abdominal pressure or pulled up by contracting adhesions within the pleural cavity will have to be determined in subsequent examinations.

Bronchoscopic Studies.—The first eight dogs in this series were examined bronchoscopically under ether anesthesia. In each case the following was noted. About 2 cm. of the bronchus remained beyond the bifurcation of the trachea and the mucous membrane appeared perfectly normal, secretions being present. The distal end of the closed bronchus was domed. Across the dome, a fine white transverse scar could be seen, around which was a network of blood vessels. Satisfactory healing of the occluded bronchus appeared to have taken place.

Blood Picture.—As said before, complete blood counts were made both before and after operation. The values for hemoglobin and erythrocyte counts varied very little, whereas there was always some increased leucocytosis postoperatively, leucocytes ranging from 2,000 to 23,000 per c.mm. of blood. In every case the increase occurred in the neutrophilic cells, and the changes occurred immediately following operation, returning to normal in about one week.

Cause of Death.—For the few dogs that died after operation the average length of life was 7.3 days. In the entire series only two animals died of pressure pneumothorax, and in both of these instances a foreign body was used in the attempt to close the bronchus; in one case a metal band was used to occlude the bronchus, and in the other a special button was used. At necropsy both band and button were found free in the pleural cavity.

COMMENT

Failure of the occluded bronchus to heal has long presented a difficult problem to those who would apply pneumonectomy and lobectomy to man. The results of our experimental work indicate that successful closure of large bronchi can be accomplished in almost every case.

Roentgenograms show conclusively that the space left on the operated side is usually totally obliterated in a few weeks. Gradual shift of the heart and mediastinal structures, as well as expansion of the remaining lung and elevation of the paralyzed diaphragm on the operated side, obliterated the dead space.

What are the changes occurring in the remaining lung? Is the result hypertrophy, hyperplasia, or in the nature of an emphysema? Sections have been made of the lungs removed at operation, and studies will be made of these and of the remaining lung after a sufficient length of time has elapsed.

SUMMARY AND CONCLUSIONS

Pneumonectomy can be successfully performed on dogs in a high percentage of cases, and that a dog with one lung can live and successfully compete with normal animals has been demonstrated.

Bronchoscopic examination reveals that perfect bronchial healing can be obtained by the method herewith outlined.

It has been demonstrated by roentgenograms that the space left by removal of a lung becomes filled by enlargement of the remaining lung and shift of the heart and mediastinum.

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SOME POSTOPERATIVE EFFECTS OF PNEUMONECTOMY

A Morphologic Study

Albert Behrend,* M.D., and Frank C. Mann,† M.D.
Rochester, Minn.

IN ANOTHER paper we reviewed the literature and described a dependable technique for experimental pneumonectomy, a method that has been singularly free of the troublesome complications most frequently encountered, namely, infection of the pleural cavity and bronchial fistula. It is the purpose of this paper to describe some of the morphologic effects of experimental pneumonectomy found on examination of dogs killed at intervals of from five days to eight years after operation.

IMMEDIATE POSTOPERATIVE EFFECTS

The operation of pneumonectomy is usually well tolerated by the dog. The animal is able to stand alone and to walk about as soon as the effects of the anesthetic have disappeared.

Dyspnea is not a prominent early symptom. The degree and the duration of dyspnea vary with the individual animal. It is usually most severe twenty-four to forty-eight hours after operation and disappears within a week thereafter. It is characterized by an increased respiratory rate and by an increase in the degree of muscular effort required to maintain respiration. Quinby and Morse observed an increase in heart rate in addition to an increase in the rate and amplitude of respiration.

Cyanosis practically never occurs in the healthy pneumonectomized animal. It is a prominent feature some time before death secondary to pleuritis with effusion or pneumonia. Therefore, it may be regarded as a prognostic sign of grave significance. Exceptionally it occurs when the animal is subjected to some extraordinary stress. For example, four puppies were delivered by one of our dogs two weeks after pneumonectomy. Dyspnea and cyanosis were marked during labor, and it appeared doubtful that the animal would survive. On the day following delivery, however, cyanosis had disappeared and the respiratory rate was normal once more.

The immediate effect of pneumonectomy on the heart, and on the right side of the heart particularly, affords an interesting problem. It must be remembered that following pneumonectomy the blood that formerly went to two lungs goes to one. An attempt was made to ob-

*Fellow in Surgery, The Mayo Foundation.
†Division of Experimental Medicine, The Mayo Foundation.
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tain electrocardiographic evidence of possible right cardiac strain or failure following pneumonectomy. Our attention, however, was called to the variability of electrocardiographic tracings on normal dogs by Katz, Soskin and Frisch; and Barnes and Mann's paper has shown that operative procedures involving the pericardium also affect the electrocardiographic tracings for the dog.

Alterations in the blood and alveolar gaseous exchange were demonstrated by Heuer and Andrus. They observed a rise in alveolar and blood carbon dioxide, and a coincident fall in alveolar and blood oxygen, for ten days after pneumonectomy, and then a gradual return of carbon dioxide and oxygen to normal in about twenty-five days. There was a decrease in oxygen saturation of the blood until the eighth day, after which there was a gradual return to normal. Andrus also found an increased blood flow, averaging 40 per cent, in the remaining lung with a return to the preoperative level within ten days. His measurements depended on the difference in carbon dioxide content of arterial and venous blood.

Thomas, Rienhoff and Allan commented in a clinical report that despite the sudden occlusion of large pulmonary vessels which the operation requires, the characteristics of sudden, accidental, pulmonary arterial occlusion (embolism) were not observed in two cases, one of an adult, the other of a child. This appears to be true also in experimental studies on animals. Mann has shown that total or almost total obstruction of the pulmonary artery or its main branches is necessary to produce sudden death of the animals, whether they be anesthetized or unanesthetized. Postmortem examination of patients who had died as a result of sudden pulmonary arterial occlusion substantiates this finding. In general it may, therefore, be stated that pulmonary emboli that kill suddenly are massive emboli.

The early and late effects of pneumonectomy on the microscopic anatomy are intimately connected and so will be discussed together.

LATER EFFECTS OF PNEUMONECTOMY

The Bony Thorax.—Permanent changes in the shape of the bony thorax were observed as early as three weeks after operation. While these changes become slightly more pronounced in the weeks that follow, the main compensatory adjustments are completed three to four weeks after a lung has been removed.

The ribs, and notably the upper ribs, tend to lose their normal concavity, with the result that there is some collapse of the thoracic wall on the side on which the lung was removed, and a resultant diminution in size of the thoracic cavity on that side. This occurred in all the animals (Fig. 1), but was most noticeable in younger dogs in which the bones were less rigid.

The Mediastinal Structures.—The heart shifts to the side of removal and further tends to obliterate the cavity left by the lung. This shift

has not appeared to be a cause of embarrassment to the dog, as it might be to man, possibly because the dog's mediastinum is generally more mobile than man's. At this point it is interesting to observe that the esophagus joins the heart and mediastinal structures in its shift toward the affected side (Fig. 1).

The Diaphragm.—No change in the position of the diaphragm has been observed after pneumonectomy except in those instances in which the phrenic nerve was sectioned. While Friedrich reported elevation of the diaphragm on the side of removal, we have not found this to occur unless adhesions were present between the diaphragm and the

Fig. 1.—Gross appearance of the thorax of a dog killed ten weeks after pneumonectomy, showing asymmetry of the thorax, marked deviation of the esophagus to the right, collapsed left lung and lack of gross emphysema, normal diaphragm and the heart in the midline. In life the heart was pushed far to the right by the left lung.

mediastinal structures; and this fact has been definitely confirmed both by roentgenographic studies and findings at necropsy (Fig. 1).

The Lymphatic Structures.—Lemon and Higgins studied the absorption of particulate matter through the normal and the paralyzed diaphragm, using a suspension of powdered graphite. The particulate matter is injected into the peritoneal cavity. By this method the lymphatic channels can be outlined clearly as they traverse the diaphragm and ascend in the thorax. They found that with the phrenic nerves intact, absorption was greater on the right side than on the left.

Section of the phrenic nerve on either side caused a diminution of absorption through that side, but even with the right phrenic nerve sectioned and the left intact, there was more absorption through the right side than the left.

The absorption of particulate matter through the intact diaphragm was studied in pneumonectomized animals. From 20 to 30 c.c. of hydrokollag was injected intraperitoneally, depending on the size of the animal, and the animal was then killed three hours after injection. It was found that even with the phrenic nerve intact, when the lung was missing on the right side, lymphatic absorption was greater on the left than on the right. It, therefore, appears that the presence of the lung is an even more important factor in the maintenance of good lymphatic circulation than is the presence of an intact phrenic nerve.

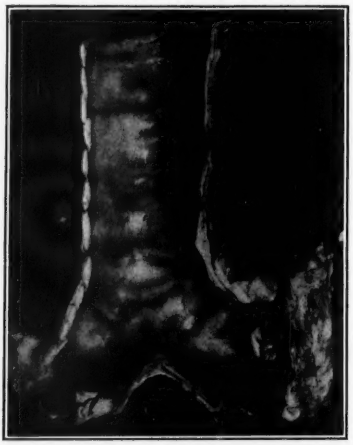

Fig. 2.—Trachea and tracheal bifurcation, showing the effective closure of the right main bronchus (four weeks after pneumonectomy).

We are unable to state whether this is due to injury to the lymphatic channels at the time of operation or to the absence of the normal respiratory movements of the intact lung.

The Bronchus.—Examination of gross and microscopic specimens of the occluded main bronchus revealed that, within one week after operation, healing was well established and that, four weeks postoperatively, occlusion was grossly and microscopically complete (Figs. 2 and 3). Our work was singularly free of the disastrous complication of pressure pneumothorax, and this we attribute to use of the technique of closure as outlined in the description of the technique of operation. The stump becomes buried in the mediastinum and oversewn pleura forms an adherent covering for the occluded end of the bronchus.

Gross Changes in the Remaining Lung.—Of paramount importance and interest are the gross and microscopic changes that occur in the

remaining lung following pneumonectomy. One is impressed by the diversity of opinions as to what does occur when a single lung remains to carry on the burden. In a measure this lack of uniformity is undoubtedly due to the unfortunate selection of the terms commonly used to describe physiologic and pathologic changes in the lung. The names "compensatory emphysema," "hypertrophy," and "hyperplasia" are frequently encountered in discussions on the effect of pneumonectomy.

"Compensatory emphysema" resembles chronic pulmonary emphysema pathologically but may be confined to small areas, as in the

Fig. 3.—Section of occluded bronchus seen in Fig. 2 (×3½).

neighborhood of an area of atelectasis. In chronic pulmonary diseases, such as pulmonary tuberculosis, its distribution may be more diffuse (Karsner). It may involve the whole lung or only a part of it. The name is unfortunate. The reduced functional capacity of a truly emphysematous lung is well established. Therefore, an emphysema cannot, in the physiologic sense, be compensatory.

Others speak of "hypertrophy" of the lung, by which we understood an increase in size due to enlargement of the individual functional unit or lobule which Miller described as comprising the alveolar ducts, the atria, air sacs and alveoli.

Still others mention "hyperplasia," by which must be meant an increase in the number of functional units, brought about by the formation of new lobules.

Hellin, for example, said that it is not clear whether there is hypertrophy, hyperplasia, or emphysema; there appears to be hypertrophy, and possibly also hyperplasia, but the picture is not that of emphysema. Friedrich said that emphysema occurs in the remaining lung. Quinby and Morse saw no emphysema. Schepelmann found emphysema, and Kawamura saw emphysema and hypertrophy but no hyperplasia on microscopic study. Möllgaard believed the alveolar spaces to be dilated

Fig. 4.—Roentgenogram made during injection of an opaque medium (thorotrast) into jugular vein, showing overexpansion of left lung and the heart pushed far to the right.

without atrophy of their partitioning walls, and that a chronic compensatory emphysema might occur depending in degree on the strength of the heart.

We can with certainty state that the enlargement of the lung which does occur is not due to emphysema. It has been our fortunate opportunity to study, both grossly and microscopically, material in which one lung had been removed at intervals of from a few days to eight years. Nor do we feel that the enlargement is due to hypertrophy of the functional unit of the lung; that is, the pulmonary lobule. Nor have we found any evidence to show that hyperplasia occurs.

Organs endowed with vital functions possess a superfluity of functional units which in time of stress, whether due to pathologic or physiologic causes, become available for use. This has been well demonstrated with regard to the kidney by Richards and Schmidt, while the enormous reserve of the liver has been demonstrated by Mann and his coworkers, to mention only two instances.

That the lung is an organ richly oversupplied with tissue capable of maintaining the respiratory function is a statement that will startle no one, but its realization is important to a proper understanding of

Fig. 5.—Trachea, tracheal bifurcation, and remaining lung removed six weeks after pneumonectomy. There is no gross evidence of emphysema.

what occurs when one lung carries on where formerly there were two. The soft crepitations caused by the opening of dormant alveoli when a deep inspiration follows a period of ordinarily quiet breathing is a phenomenon of common clinical experience. Furthermore, the average tidal air in man is estimated at from 400 to 500 c.c., while vital capacity is roughly seven times as great (Wright). Finally, Willius stated that the oxygen consumption of a normal individual may increase ten- or twelve-fold during exercise. The removal of one lung results in a sudden demand for increased work on the lung that remains. This demand is met either by an increase in the number of functional units

or pulmonary lobules in use at one time or, as Rienhoff and his associates have recently suggested, by simple dilatation of the terminal portion of the respiratory lobule. It is highly probable that both factors contribute to cause increased expansion of the lung.

There can be little doubt that the remaining lung following pneumonectomy enlarges and, as a rule, increases in weight. By means of roentgenograms and the injection of an opaque medium (thorotrast) into the jugular vein, we were able to show the outline of the left lung entirely filling its own side of the thoracic cage and the apical portion of the right thorax. The heart in such cases is displaced far to the right (Fig. 4). We know that this is a true displacement and not one due to adhesions drawing the heart to the right because, when the animals are killed, the left lung collapses on opening the thorax and the heart is found lying in its usual midthoracic position. Furthermore, the lung does not present the gross picture of emphysema, whether the specimen is seen days or years after operation (Fig. 5).

In most instances following pneumonectomy the lung that remains increases in weight (Table I). The expected weight of the remaining

TABLE I

INCREASE IN WEIGHT OF REMAINING LUNG FOLLOWING PNEUMONECTOMY

WEIGHT OF DOG, KG.	WEIGHT OF RIGHT LUNG, GM.	ESTIMATED WEIGHT OF LEFT LUNG, GM.	TIME FOLLOWING OPERATION WHEN DOG WAS KILLED	WEIGHT OF LEFT LUNG, GM.	INCREASE, PER CENT
6.0	41	29	5 days	39.5	72
10.0	68	48	12 days	57.5	83
8.8	69	50	21 days	51.0	
8.7	55	39	5 weeks	59.5	69
9.3	56.5	39	10 weeks	56.0	70

lung under normal conditions was estimated in the following manner. The right and left lungs of a series of normal animals killed for other purposes were weighed, and it was found that the average weight of the left lung was 30 per cent less than that of the right. In our series of animals the right lung was always removed at operation and weighed, and the weight of the left lung was estimated from this figure. The increase in weight of the left lung was then estimated from this latter figure. The increase in weight of the left lung appears to be due to two factors; namely, to an increase in the amount of blood in the single lung, and to proliferation of histiocytes in focal areas, as will be described in the discussion of the microscopic changes occurring postoperatively.

MICROSCOPIC CHANGES FOLLOWING PNEUMONECTOMY

It is important in any microscopic study of the abnormal lung to appreciate the extremely variable picture presented by the normal lung. Several factors may contribute to the production of this variability,

such as sharpness of knives, the amount of handling of the tissue, the fixative used, and so forth. Furthermore, the conditions under which the animal has lived can influence the microscopic appearance. Thus while the control lung will in the main be normal, it is not difficult to find areas in which the alveoli are collapsed and others in which the alveoli are decidedly dilated, even to a degree that suggests emphysema (Fig. 6). It may even be possible to find normal, collapsed, and dilated alveoli in the same slide, so that special caution must be exercised in the study of experimental material.

At the time of operation, blocks were cut from all the lobes of the removed lung, and these were fixed in 10 per cent formalin, embedded

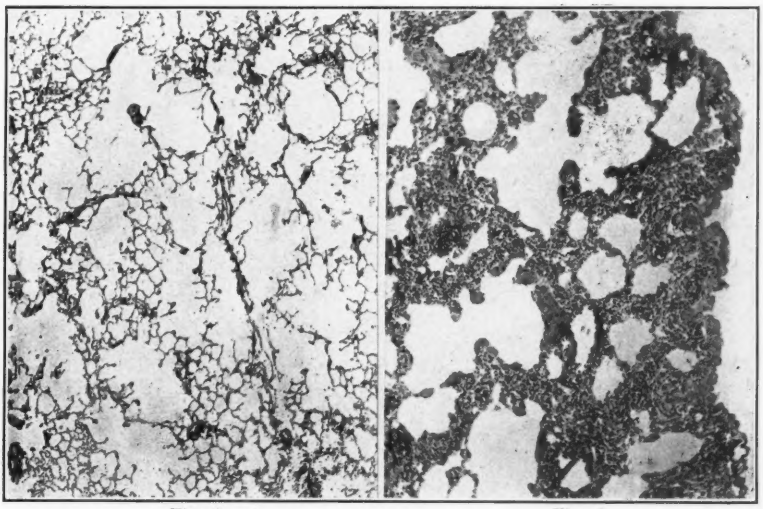

Fig. 6. Fig. 7.

Fig. 6.—Section of control lung removed at operation, showing apparent tendency to emphysema. Such a picture in the postoperative specimen might be interpreted as an effect of the operation (×40).

Fig. 7.—Proliferation of histiocytes seen in focal areas throughout the remaining lung. This specimen is from an animal killed ten weeks after pneumonectomy.

in paraffin, and sections were stained with hematoxylin and eosin. Selected sections were studied using the van Gieson and Mallory-Heidenhain stains for connective tissue and the Bielschowsky silver stain for reticulum fibers.

Microscopic study was then made of sections of the remaining lung of animals that had been killed at intervals from five days to eight years after pneumonectomy. In no instance was evidence found to suggest the presence of a generalized emphysema, or hypertrophy and hyperplasia of the pulmonary lobules. For the most part, the size and the thickness of the blood vessels were not materially changed, although

some sections from animals killed about one week after operation revealed an apparent increase in the number of smaller vessels distended with blood.

The remaining lung appeared remarkably like the normal with one exception. In almost every animal there were focal areas showing decided proliferations of histiocytes (Fig. 7). This proliferation manifested itself as a symmetrical thickening of the normal alveolar walls. Usually it was most pronounced in the region of the bronchi. Cellular infiltration about the bronchus was commonly seen in the normal lung, but many of the cells resembled large lymphocytes rather than histiocytes.

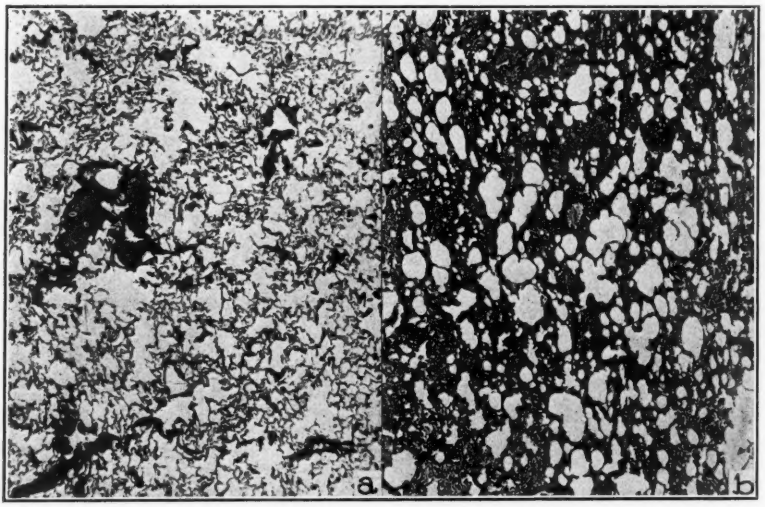

Fig. 8 *a* and *b*.—Sections of the lungs of two animals killed eight years after pneumonectomy, showing the widely different pictures. In *b*, the cells of the focal area of histiocytosis have become fibrocytes (×35).

It was possible to follow the progressive changes occurring in these cells with the passage of time. A histiologic study of the remaining lung secured a few days after pneumonectomy revealed the histiocytes as large cells with granular cytoplasm and a large, rather pale-staining nucleus. With the Bielschowsky stain these cells are not visualized at all, and one can see only the skeleton of the alveolar wall in its reticulum fibers. Later after pneumonectomy the cell had become smaller and the nuclei shrunken and stained more darkly. In the eighth year following pneumonectomy the histiocytes appeared to have become transformed into fibrocytes (Fig. 8 *a* and *b*).

Coincident with this "aging" process which the new histiocytes undergo occurs an increase in the capillary network of the remaining

lung, which begins about six weeks after pneumonectomy has been performed. The new capillaries from the focal areas of cellular proliferation intertwine among these cells, largely increasing the blood which can circulate through the lung. It is possibly significant that these focal areas of cellular proliferation are most prominent in the neighborhood of the terminal bronchi and of the pulmonary vessels that accompany the bronchi. It seems highly probable that the increase in the capillary bed acts as an important compensatory factor following pneumonectomy.

This development of new vessels has been studied in the material obtained from animals killed at various intervals after pneumonectomy. While new vessels are seen in the lungs of animals killed five days

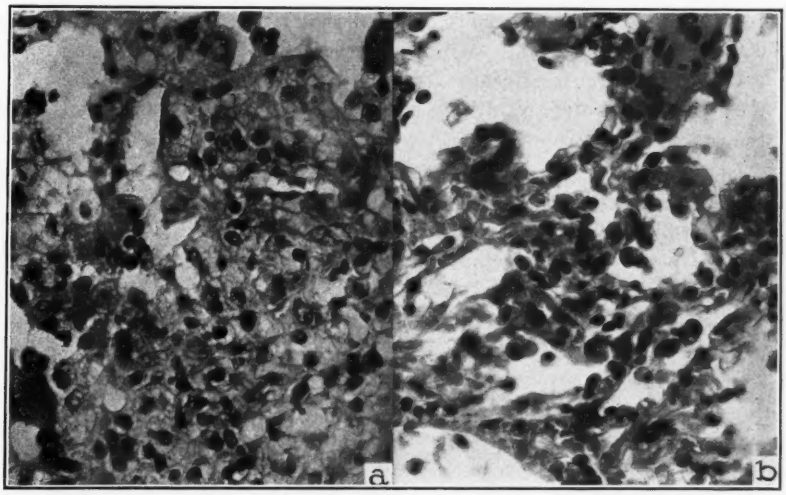

Fig. 9.—Comparable areas from the alveolar wall in an animal five days after pneumonectomy (*a*) and eight years after pneumonectomy (*b*). Note the large histiocytic cells on the left, with foamy cytoplasm and granular nuclei, compared to the smaller dark-staining nuclei and diminished amount of cytoplasm on the right. Note also the absence of capillary blood vessels on the left and their presence on the right (×450).

after pneumonectomy, the phenomenon is usually strikingly apparent in the remaining lung of animals killed later (Fig. 9). In animals killed later (Fig. 9 *a* and *b*) moderately increased fibrosis is demonstrated by the van Gieson and Mallory-Heidenhain stains, and the increase in the reticulum framework of the alveolus is strikingly demonstrated by the Bielschowsky stain.

SUMMARY AND CONCLUSIONS

Pneumonectomy is well tolerated by the dog.

Dyspnea and cyanosis are uncommon and, when they occur, are due to complications.

Following pneumonectomy the heart is pushed toward the operated side by the increase in size of the remaining lung. The bony thorax on the side of removal becomes smaller. The diaphragm is unaffected. The main lymphatic channels on the side of removal become less active. The occluded bronchus is grossly and microscopically closed four weeks after operation.

The remaining lung enlarges and becomes heavier. The enlargement is not due to hypertrophy, hyperplasia, or emphysema, but is due to an increase in the number of pulmonary lobules functioning at a given time or to simple dilatation of the air cells, or both. The increase in weight of the remaining lung is probably due to the added blood in this one lung and to an increase in the number of histiocytes.

Microscopically, hypertrophy, hyperplasia, emphysema, or even simple dilatation, is not generally evident in the alveoli of the single lung remaining after pneumonectomy. Focal areas of histiocytic proliferation have been noted, resulting in thickening of the alveolar walls. In the course of years these histiocytes acquire the characteristics of fibrocytic cells.

New capillaries were found to permeate the focal areas of histiocytic proliferation, beginning about six weeks after pneumonectomy. This is possibly an important factor in the compensatory adjustment after pneumonectomy.

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DISCUSSION

DR. POL N. CORYLLOS.—It really is of great significance whether the contralateral lung, after pneumonectomy, presents simple emphysema and dilation or real hypertrophy with hyperplasia of its anatomical respiratory units.

I have been deeply interested in this problem. As Dr. Behrend, I have no definite facts to present to you. I believe, however, that there is another way of attacking this problem with some chances of success. This procedure is based upon the knowledge that in polycystic lung with secondary bronchiectasis, or in what we call congenital bronchiectasis, the distinction between congenital and acquired cysts may be made by the presence or absence of carbon infiltration of these parts of the lung. We could thus attack the problem by submitting dogs to long sojourns in an atmosphere saturated with lamp black. Pneumonectomy could then be performed upon these dogs, and the animals could be sacrificed at varied intervals. Macroscopical and especially microscopical examination of the lung may help in distinguishing strands of new-formed pulmonary tissue, if there is any.

DR. RALPH B. BETTMAN.—Many years ago, I believe it was in Rochester, I presented a report of experiments I had made on pneumonectomy on dogs. In this series most of the dogs died because of reopening of the stump, and the conclusion reached by studying these and the few dogs which survived was that the bronchial stump itself has practically no inherent recuperative powers and that closure of a bronchial stump could occur only by the result of the ingrowth of extraneous tissue. I want to know whether Dr. Behrend examined the closed bronchial stumps and whether or not he can tell us how large a rôle the pleura had in effecting the closure.

DR. ALBERT BEHREND.—As far as I can tell, the manner in which these bronchi heal is by fibrous union, and I felt that an important point in the healing was to supply sufficient blood to the terminal portion of the bronchus so that healing could take place under normal conditions. The pleural surface sewn over the occluded bronchus becomes intimately fused with it and is a further aid in obtaining permanent closure.

EMPYEMA: A METHOD OF TREATMENT WITH CONTINUOUS IRRIGATION AND DRAINAGE

ALLAN PENNY BLOXSOM, M.D., HOUSTON, TEXAS

THE present device relates to a method especially useful in conjunction with treatment of empyemas requiring irrigation and drainage, and has for its principal object provision of a simple means for continuous irrigation and drainage of an empyema cavity.

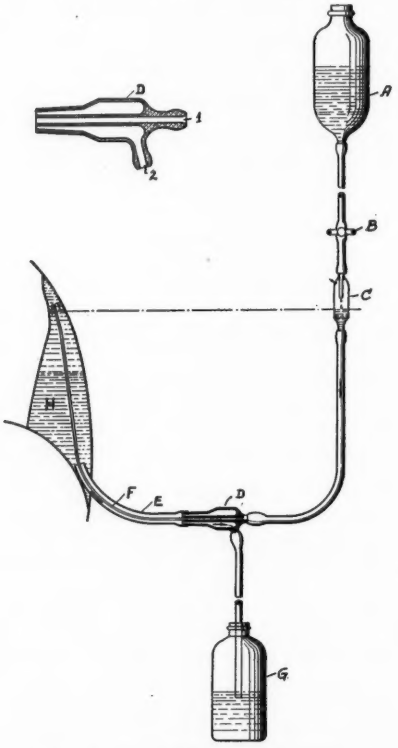


Fig. 1.—Illustration of apparatus.

Another object of the device is the elimination of the present un satisfactory method of alternately allowing fluid to run into a cavity, expanding that cavity, and then allowing fluid to drain from the cavity.

A still further object of the device is the elimination of the danger of shifting the mediastinum through the ability of the device to main-

tain as desired, either a negative pressure, or a positive pressure, with continuous irrigation and drainage.

The accompanying illustration (Fig. 1) reveals the apparatus assembled into a unit suitable for continuous irrigation and drainage. *A* is a gravity bottle connected with a Murphy drip tube, *C; B* is a valve for adjusting the rate of flow of the irrigating fluid into the bowl of the Murphy drip tube, *C*. The arrow at the top of that Murphy drip tube, *C*, indicates an opening to the outside, so that pressure cannot build up above the level of the irrigating fluid in the Murphy drip tube. Rubber tubing connects the Murphy drip tube to the intake nozzle, *1*, of a glass return flow ear douche bulb, *D*. A No. 8 catheter, *F*, is drawn through the inner glass tube, *1*, of the return flow bulb, *D*, by means of a needle and thread. This catheter is prevented from slipping through the inner glass tubing of the bulb, *D*, by the dilated end. This also insures a tight fit so that fluid does not leak around the intake tube. A No. 24 catheter, *E*, is inserted just into the empyema cavity, *H*, by means of a cannula and trocar. The end of the No. 24 catheter is then slipped over the large intake opening of the glass douche bulb, *D*, and the No. 8 inlet catheter is pushed through the inner glass tubing, *1*, until it passes far into the empyema cavity. A rubber tubing connects with the outlet opening, *2*, of the glass douche bulb and extends down below the water level of a water bottle, *G*, that is conveniently attached to the side of the bed of the patient.

To maintain a negative pressure in the empyema cavity, the water bottle, *G*, must be below the level of the empyema cavity, and the Murphy drip tube, *C*, slightly below the level of the empyema cavity.

In order to maintain a positive pressure in the empyema cavity, the water bottle, *G*, must be either at the level or above the fluid cavity, *H*, to be drained and irrigated, and the Murphy drip tube, *C*, must be slightly higher than the level of the water in the water bottle, *G*.

REPORT OF CASE

K. W., a white male, aged nineteen months, was first seen March 9, 1936, at which time the father stated that a week previously the child had had a cold. On March 6, the child suddenly developed fever of 104° and began to breathe rapidly. This temperature continued high, and on the third day he became so acutely ill that the parents thought it necessary to call a physician. He was seen at home, and the positive physical findings were fever of 104°; respiration 64; pulse 148; slight cyanosis of the lips; some thin serous discharge from the nose; moderate congestion in the throat; very slight dullness over the left upper lobe anteriorly and posteriorly; and diminished tubular breath sounds in this area. In addition, there was a tremendous abdominal distention. A diagnosis of upper left lobar pneumonia was made and hospitalization was advised.

Patient was admitted to the St. Joseph Hospital on March 9, 1936. On admission his temperature was 104.6°, and a short time thereafter his temperature increased to 105.2°. His pulse was 160. He was placed in an oxygen box, and the concentra-

Fig. 2.—Massive pleural effusion of the left side, showing displacement of the heart to the right.

Fig. 3.—Taken two days after the tubes were inserted into the chest cavity. The smaller catheter extends into the lower part of the chest cavity, and has its tip just above the diaphragm. The outer casing extends a little too far into the chest cavity.

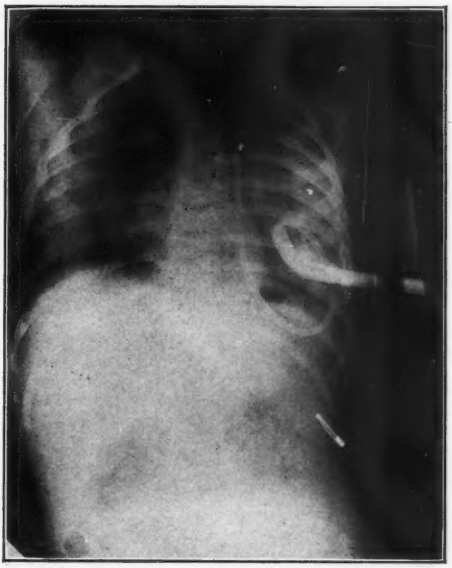


Fig. 4.—Taken after air has leaked into the chest cavity around the outer casing. The intake tube extends into the upper part of the chest cavity. The outer casing is in the correct distance (about one-half inch above the bend).

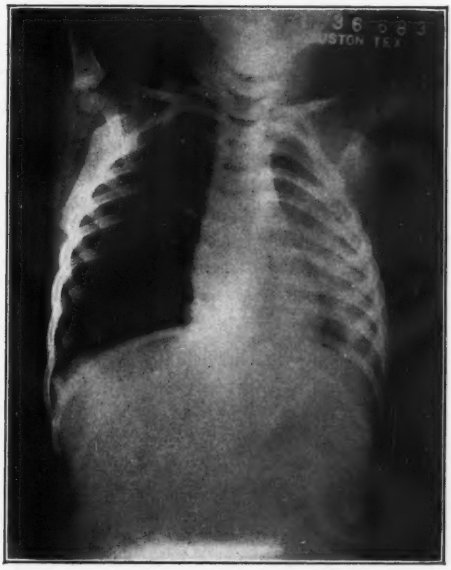


Fig. 5.—Taken the day the tubes were removed from the chest cavity, after being in place twenty-one days. The lung is expanding and some air remains in the chest cavity. There is some thickening of the pleural surfaces.

tion of oxygen kept between 75 and 80 per cent. A blood examination at this time showed a white blood count of 12,000, with 70 per cent polynuclear neutrophiles and 30 per cent small lymphocytes, and a hemoglobin reading of 83 per cent. A urinalysis showed a faint trace of albumin, no sugar, and a few hyalin and finely granular casts. An x-ray examination made on admission, showed a bilateral peribronchial infiltration of the lungs with the infiltration being most pronounced in the upper lobe of the left lung. There was no evidence of free fluid in the pleural cavities. Another blood examination made on March 12 showed a white blood count of 12,200 with 64 per cent polynuclear neutrophiles and 35 per cent small lymphocytes.

The child appeared to be doing so poorly on March 12 that he was given 200 c.c. of whole blood from his mother. The following morning, March 13, the entire left side of the child's chest was flat, and no breath sounds could be elicited. A diagnosis

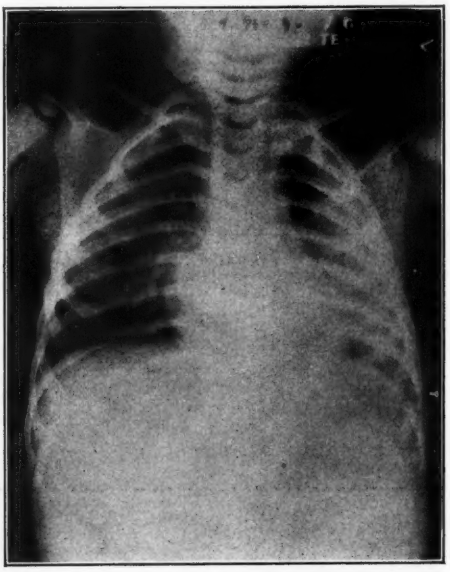


Fig. 6.—Taken two weeks after irrigating and drainage tubes were removed. Physical examination at this time revealed breath sounds being slightly diminished over the left lower lobe.

of pleural effusion was made and x-ray examination (Fig. 2) confirmed the diagnosis and showed the mediastinum to be displaced to the right. The chest was tapped and 60 c.c. of thin pus was obtained. A twenty-four-hour culture showed a good growth of staphylococci.

The child's respirations were considerably better after the initial tap. He continued to have a great deal of distention, and the following morning, March 14, 170 c.c. of fluid, more purulent, were removed from the chest cavity. The next day the child was considerably relieved when 175 c.c. of chest fluid were removed. On March 16, 55 c.c. of fluid were removed. X-ray examinations on this date showed the density of the left chest to be approximately the same as at the last examination, and the mediastinal displacement continued to be present.

On March 17, 120 c.c. of pus were aspirated from the chest and a short, rubber No. 24 catheter was inserted in the left seventh interspace slightly medial to the angle

of the scapula. The continuous irrigation and drainage tubes were then attached and normal saline allowed to irrigate the chest cavity, keeping the chest cavity under negative pressure.

The child for the first time breathed well and had a good day. His temperature dropped to 102.2°. He was brighter and seemed more interested in his food.

The rate of flow of the irrigating fluid was placed at 2,000 c.c. for the twenty-four hours. Twelve hours after the tube was placed in the chest wall, the outlet tube became stopped up from a large clot of pus, evidently due to an insufficient amount of irrigating fluid to keep the pus in sufficiently small pieces to enter between the inner and outer tubes. This was easily remedied by moving the inner tube and allowing it to act as a stilette, and for the remainder of the time the tubes were in the empyema cavity, there was no blockage (Fig. 3).

The child continued to do well, his temperature remaining at 100° to 101°; his respirations were easy, and he was in no way toxic. On March 25, his hemoglobin was 85 per cent; red blood count 4,860,000; white blood count 14,600; polymorphonuclears 42 per cent, and polynuclear neutrophiles 56 per cent.

On March 22, because of the outer casing being too far in the chest cavity, it was pulled out approximately one inch, and the inner tube about two inches. During the night, air escaped into the empyema cavity around the outside of the large casing (Fig. 4).

The inner intake tube was gradually withdrawn from the chest cavity, and the larger outer drainage tubes were removed on April 4, 1936 (Fig. 5).

There was little or no drainage from the wound in the chest after removal of the tubes, and the child was allowed to go home on April 7. The chest wound healed completely in six days.

A follow-up examination (Fig. 6) made on April 21, revealed the breath sounds somewhat diminished in this area. The child was eating well, walking, and had not had fever at any time since leaving the hospital.

SUMMARY

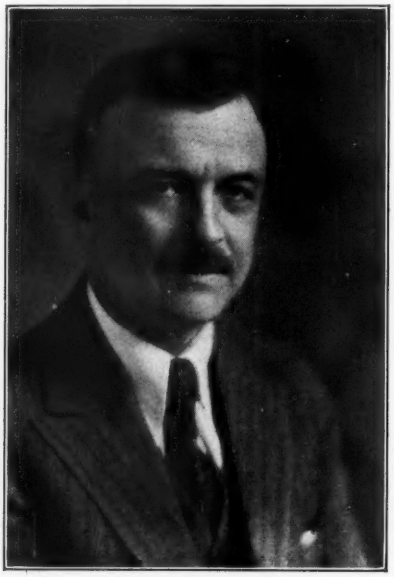
This method of treating empyema was successful in the above case and is recommended for the following reasons:

1. Irrigation and drainage can be made continuous.
2. Irrigation and drainage, because of the negative pressure maintained in the empyema cavity, can be instituted early without danger of tearing adhesions in the empyema cavity and shifting the mediastinum.
3. Cleansing of the pleural cavity was thoroughly accomplished, so that little thickening of the pleural surfaces resulted.
4. Operation of the device is extremely simple.

In Memoriam

HOWARD LOMBARD BEYE
1886—1936

The tragic and untimely death of Howard Lombard Beye, Professor of Surgery at the University of Iowa, was a great loss to the surgical profession. He was killed in an automobile accident near Marshalltown, Iowa, on September 29, 1936, while motoring to Sioux City, Iowa, where he was to have taken part in a medical meeting on tuberculosis.



Howard L. Beye

Dr. Beye was born in Oak Park, Illinois, September 24, 1886. He was graduated from the University of Wisconsin with the degree of Bachelor of Science in 1909 and received his medical training at Rush Medical College, graduating in 1911. Following a year and a half of interneship at the Cook County Hospital, he was made Assistant in Medicine at Rush Medical College. He accompanied his friend and teacher, Dr. Charles Rowan, to the University of Iowa in 1914 where he was appointed Instructor in the Surgical Department. In 1924 he was made Professor of Surgery, and three years later, succeeded Dr. Rowan as chairman of the department.

During the World War he was wounded in action at Château-Thierry. He was married to Ruth Ketcham of Oak Park on his return home. This happy union was blessed with six children: Helen, Cyrus Lombard, Jane Waters, Barbara, Charles Rowan, and Ruth.

Although his contribution to the surgical literature included many subjects in general surgery, Dr. Beye's chief interest more recently had been in the field of thoracic surgery. The worth of his contributions is reflected in the wide recognition given them as evidenced by his membership in the leading societies. These included the Johnson County Medical Society, the Iowa State Medical Society, the American Medical Association, the American College of Surgeons, the Western Surgical Association, the American Association for Thoracic Surgery, and the American Surgical Association. His contributions to the surgical literature during the past twenty years included problems dealing with the biliary system, osteomyelitis, bone sarcoma, empyema, fractures, and bronchiectasis.

Dr. Beye was well known throughout the states, but more especially in the state of Iowa, where many have had the good fortune to attend his undergraduate or postgraduate clinics and lectures, which were always given in a clear, concise, and impressive manner. He was an outstanding teacher and his ability both to instruct and stimulate made him an outstanding medical educator.

Dr. Beye will always be remembered as a shining example of honesty and straightforwardness. His ambition was to increase his knowledge in the diagnosis and treatment of surgical diseases and to impart successfully that knowledge to his students, both graduate and undergraduate. He spent innumerable hours in study in the laboratory devoting his life to his work in order that he might realize this goal.

W. E. Adams.

Department of Reviews and Abstracts

Conducted by Duff S. Allen, M.D., Associate Editor

Selected Abstracts

Lung Abscess in Children. Frank P. Mathews. Arch. Pediat. **54**: 1, 1937.

The increasing frequency of pulmonary suppuration in children is shown by a comparison of reported cases in the literature. The author believes that "wholesale" removal of tonsils and increasing accuracy of diagnosis of chest disease account for the increase.

Analysis of the cases of lung abscess occurring in children between the ages of two and ten at the St. Luke's Hospital, New York, shows that lung abscess in children is more amenable to treatment than in adults. This finding is in agreement with other and larger series in the literature.

The right lung was involved more often than the left; the lower lobes more frequently the site of the abscess. The author considers these observations give strong support to the aspiration theory of invasion in childhood.

Cultures and smears of the pus obtained revealed mixed infections, but fusiform bacilli and spirochetes which are often seen in adult cases were not found. The cases were not conspicuously malodorous as compared to adult cases.

The author believes lung abscess in children should be treated medically for six weeks. If progress is stationary, bronchoscopy and open operation may be done. The two-stage operation is favored. Artificial pneumothorax and operations on the phrenic nerve are not considered safe or suitable.

Prognosis is favorable as compared to the prognosis in adults with lung abscess.

Brian Blades.

Spontaneous Hemopneumothorax. Henry U. Hopkins. Am. J. M. Sc. **193**: 763, 1937.

Three cases of nontraumatic hemopneumothorax are described and forty-six cases in the literature summarized. The author believes the condition more common than recorded cases indicate.

The cases are divided into two groups: those patients having tuberculosis, and a second and much larger group, those without demonstrable tuberculosis.

Two possible sources of hemorrhage are discussed: the rupture of an emphysematous subpleural air vesicle with tearing of a blood vessel in the wall of the vesicle, or bleeding from preexisting pleural adhesions which are torn. The second source of hemorrhage seems more likely from autopsy findings.

Spontaneous hemopneumothorax occurs most frequently in patients who have previously been in good health. Pain is usually localized but may involve the entire side of the chest and radiate to the abdomen. Dyspnea may be slight or extreme. Systemic signs of hemorrhage may be slow or never appear. If the bleeding is severe, signs of shock with pain in the upper abdomen may lead to an incorrect diagnosis of "surgical abdomen."

Treatment depends on the severity of shock and the amount of bleeding. Aspiration is justifiable for control of dyspnea during the first forty-eight hours, but not as a routine measure. When the bleeding has ceased, the fluid may be aspirated and replaced by air.

Brian Blades.

A Preliminary Note on Sympathetic Shock in Thoracoplasty. F. Ronald Edwards. Brit. J. Tuberc. **31**: 7, 1937.

An unusual form of shock following the exposure and removal of the transverse processes of the upper dorsal vertebrae is described. Bleeding from the large venous plexus formed by extraspinal and intraspinal anastomosis, necessitating probing of the tissues to locate the bleeding point, makes the shock more pronounced.

The rapidity of onset is characteristic. Extreme collapse may occur in two or three minutes. Respirations become rapid; cyanosis may appear; blood pressure falls, and the pulse is rapid and of poor volume.

The author believes that this particular form of shock is caused by the stimulation of the sympathetic nervous system. The inferior cervical ganglion and upper two dorsal ganglia are in direct relation to the anterior surfaces of the heads of the upper three ribs. The position may vary from just above the neck of the first rib to the upper border of the third rib.

A technique to infiltrate the inferior cervical and upper two dorsal ganglia with novocaine after exposure of the ribs is described.

Ten cases have been so treated during the performance of thoracoplasty operations. There was complete absence of shock. The author concludes that the procedure is of definite value.

Brian Blades.

Intensive Collapse Therapy in Pulmonary Tuberculosis. G. I. Leslie and R. S. Anderson. Am. J. M. Sc. **193**: 149, 1937.

The detailed results of collapse therapy for pulmonary tuberculosis in a group of 1,124 patients is described.

Seventy-eight and eight-tenths per cent of the 1,124 patients received some form of collapse therapy.

Eight hundred and twenty-three patients are now discharged. Five hundred ninety-five of the discharged patients had collapse therapy.

The following results were obtained:

	Discharged Patients	Discharged Patients With Collapse Therapy
Arrested Cases	47.3%	55.4%
Favorable Results	67.1%	77.8%
Cavity Closure	57.0%	71.3%
Closure and Decrease in Size of Cavity	69.6%	84.2%
Sputum Conversion in Positive Cases	59.0%	72.7%
Negative Sputum	70.7%	79.3%

Corresponding data for the entire group of 1,124 for cavity closure and sputum conversion in positive cases were slightly better than those of the discharged group alone.

A comparison of the results in sanatoriums where collapse therapy is employed on relatively fewer patients is given.

The authors consider their results justify the policy of intensive collapse therapy in about three-fourths of patients with adult type of tuberculosis.

Brian Blades.

Phrenic Interruption Combined With Artificial Pneumothorax for Pulmonary Tuberculosis. Fred R. Harper. Am. Rev. Tuberc. **35**: 475, 1937.

A group of 44 cases treated by a combination of phrenic interruption and artificial pneumothorax is analyzed. All of these patients were observed for at least a year. In 16 of the 44 cases, phrenic interruption was first done, while in 28 instances, pneumothorax was the first treatment.

Following the classification of the National Tuberculosis Association, the 44 cases were divided as follows: 24 far advanced; 16 moderately advanced; 3 minimal; one, pneumoconiosis with superimposed tuberculosis. Thirty-one cases had bilateral lesions, 9 cases bilateral cavitation. Thirty-seven of the 44 cases had sputum containing tubercle bacilli. The average age was thirty-eight years for the group. The ages varied from twenty-three to fifty-six years.

Seventeen of the cases were considered cured following combined phrenic interruption and pneumothorax. Four cases are classified as

arrested. Fourteen are apparently arrested; the time interval has not been long enough to call them arrested cases. Six patients required further collapse procedures. Three patients died.

The combination of the two procedures was effective in closing 80 per cent of the cavities that had failed to close with phrenic interruption or pneumothorax alone.

The combination of the two methods of collapse caused the sputum to become negative for tubercle bacilli in 89 per cent of the cases. In instances of bilateral cavitation, the cavities became completely closed in 77 per cent of the cases.

The interval between pneumothorax refills could be increased following phrenic interruption. The incidence and severity of pleural effusion seemed less when the two procedures were combined.

The author considers the results obtained by the combination of pneumothorax and phrenic interruption so gratifying that the use of the second operation is indicated in a considerable number of patients in which either procedure used alone proves ineffective.

Brian Blades.

The American Association for Thoracic Surgery

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The American Association for Thoracic Surgery

The twentieth annual meeting of the American Association for Thoracic Surgery was held at Saranac Lake, N. Y., on May 31, June 1, 2, with a very large attendance.

Professor Hans Christian Jacobaeus of Stockholm, Sweden, was the guest speaker. The subject of his address was, "Bronchospirometry and Its Use in Determining Indications for Thoracoplasty in Bilateral Pulmonary Tuberculosis."

Dr. Stuart W. Harrington of Rochester, Minnesota, was elected President for the current year; Dr. Harold Brunn of San Francisco was elected Vice President; Drs. Graham, Churchill, and Meade were reelected to their respective offices; Dr. Dan C. Elkin of Atlanta was elected to the Council.

As a memorial to Dr. Willy Meyer, the founder of the American Association for Thoracic Surgery, a gavel was presented to the Association by his son, Dr. Herbert Willy Meyer.

The amendments to the Constitution which were finally passed at this meeting deleted Art. IV, Sec. 1, which read: "As a component member of the Congress of American Physicians and Surgeons, this Association subscribes to the Constitution and By-Laws of the Congress." Also Art. VIII, Sec. 1, was changed to read: "The time, place, duration and procedure of the annual meeting of the Association shall be determined by the Council, and the provisions of the By-Laws."

The following amendments to the By-Laws were adopted and became at once effective.

Article IV

SEC. 1.—Candidates for membership in this Association must be formally nominated and seconded, in an approved manner, by not less than three Active or Senior Members. Such nomination must have been in the hands of the Membership Com-

mittee for not less than four months, and the name of the candidate must have been published to the Association as a whole before final action may be taken on any new candidate for election to Active or Associate Membership. Provided the foregoing requirements have been met, and the candidates have been approved by the Membership Committee and by the Council, their names shall be presented to the Association at a regularly convened annual meeting for final action. A three-fourths vote of those present and voting shall be required to elect. Any candidate for membership in this Association who has failed of election for two successive years, shall automatically cease to be a candidate and may not be renominated until after a lapse of three years.

SEC. 6.—The report of the Membership Committee shall be rendered at the annual executive session of the Association. Candidates shall be presented in groups in the following order: Candidates for Honorary Membership; retirement of Active Members to Senior Membership; Associate Members eligible to Active Membership; candidates for Active Membership; candidates for Associate Membership.

SEC. 7.—The Council shall recommend that any member whose dues are in arrears for two years, or who has been absent, without sufficient excuse, from four consecutive annual meetings, shall have his membership terminated.

Article VII

SEC. 2.—Annual dues for Active Members shall be $10.

SEC. 3.—Annual dues for Associate Members shall be $3.

SEC. 4.—Annual dues for Senior Members shall be $3.

SEC. 9.—Associate and Active Members must subscribe to the Journal of Thoracic Surgery to retain their membership status.

SEC. 10.—Senior Members may retain their membership status by the payment of annual dues, but subscription to the Journal of Thoracic Surgery is optional.

(Note:—Bills for membership dues and for subscriptions to the Journal of Thoracic Surgery will be mailed to members by the Treasurer after the annual meeting.)

The report of the Committee on Certification of Thoracic Surgeons was read by the Chairman, Dr. Carl Eggers. The resolutions adopted by the Committee were approved by the Association. They read as follows:

1. Be it resolved that the American Association for Thoracic Surgery recognize the American Board of Surgery as the parent organization which should properly be in control of all matters pertaining to the training and certification of surgeons and surgical specialists.

2. Be it further resolved that the American Association for Thoracic Surgery signify to the American Board of Surgery its willingness to cooperate with it, if and when the special certification of thoracic surgeons seems desirable.

3. Be it further resolved that in case a subcommittee be desired by the American Board of Surgery it be appointed by the Council of the American Association for Thoracic Surgery and that it be composed of surgical members of the Association.

4. The functions of the subcommittee should be determined by the American Board of Surgery.

In recognition of their outstanding contributions to the field of thoracic surgery, the following men were elected to Honorary Membership:

Professor Hans C. Jacobaeus, Stockholm, Sweden
Dr. Nathan W. Green, New Canaan, Conn.
Dr. Edward W. Archibald, Montreal, Canada

The following Active Members were retired to Senior Membership at their own request:

Dr. Edward W. Peterson, New York, N. Y.
Dr. Alfred Bazin, Montreal, Canada

The following Associate Members, in recognition of their contributions to thoracic surgery, were elected to Active Membership:

Dr. Willard VanHazel, Chicago, Illinois
Dr. Howard H. Bradshaw, Philadelphia, Pa.
Dr. W. L. Rogers, San Francisco, Calif.
Dr. George M. Curtis, Columbus, Ohio
Dr. John H. Gibbon, Jr., Philadelphia, Pa.
Dr. Albert O. Singleton, Galveston, Texas

The following candidates were elected to Associate Membership:

Dr. Thomas B. Aycock, Baltimore, Md.
Dr. William W. Buckingham, Kansas City, Mo.
Dr. David A. Cooper, Philadelphia, Pa.
Dr. Paul D. Crimm, Evansville, Ind.
Dr. Edgar W. Davis, Washington, D. C.
Dr. George Deshaies, Montreal, Canada
Dr. Richard H. Dieffenbach, Newark, N. J.
Dr. Edward P. Eglee, New York, N. Y.
Dr. Frederick R. Harper, Tucson, Arizona
Dr. John S. Harter, Sanatorium, Miss.
Dr. Minas Joannides, Chicago, Illinois
Dr. Henry W. Leetch, Saranac Lake, N. Y.
Dr. Gustaf E. Lindskog, New Haven, Conn.
Dr. Herbert Meltzer, Ninette, Manitoba, Canada
Dr. Max Pinner, Ithaca, N. Y.
Dr. Oscar S. Proctor, Seattle, Wash.
Dr. H. McLeod Riggins, New York, N. Y.
Dr. Paul C. Samson, Oakland, Calif.
Dr. Vernon D. Schaffner, Kentville, Nova Scotia
Dr. H. Brodie Stephens, San Francisco, Calif.
Dr. Robert Glen Urquhart, Norwich, Conn.

The following committees were appointed:

Membership Committee

Dr. Ethan F. Butler, Chairman, Ithaca, N. Y.
Dr. I. A. Bigger, Richmond, Va. — Dr. Claude S. Beck, Cleveland
Dr. Emile Holman, San Francisco — Dr. Fraser B. Gurd, Montreal

Program Committee

Dr. Frank Boland, Atlanta, Ga. — Dr. Dan C. Elkin, Atlanta, Ga.
President, Secretary, Editor, ex officio.

The next annual meeting of the Association will be held in Atlanta, Georgia, during April, 1938. The headquarters will be at the Atlanta Biltmore Hotel.

The closing date for submitting applications for places on the program at the next meeting will be February 1. Intending contributors are requested to send comprehensive abstracts of their papers to the Secretary, Dr. Richard H. Meade, Jr., 2116 Pine St., Philadelphia, as early as possible.

In accordance with the amendment to the By-Laws dealing with the nomination of new members (Art. IV, Sec. 1), the closing date for the year will be December 1, 1937. Members wishing to nominate candidates must secure the proper forms from Dr. Ethan Butler, Chairman of the Membership Committee, Biggs Memorial Hospital, Ithaca, N. Y., and return them to him before that date.

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